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Herd Eradication of Str. agalactiae by Simultaneous Treatment of All Cows with 5 Doses of 100,000 units of Penicillin at Daily Intervals and Disinfection

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It has long been known that *Str. agalactiae* free herds can be established by segregation or, better, immediate removal of animals shown to be infected by means of cultural milk tests and gradual replacement by clean animals, and that these herds can be maintained free (Minett and others, 1933; Stableforth and others, 1935; Plastridge and others, 1936, 1939, 1942 and 1946; Hastings, 1937, 1943; Marval, 1939; Little, 1940; Mattick and others, 1941; Cunningham and others, 1947). It is also known that free herds can quickly be built up from tested heifers, because nearly all are free when they first calve (Stableforth, 1935, 1938; Hastings and others, 1937, 1940; Plastridge, 1934, 1942; van Rensburg, 1942). In most herds, however, disposal of infected animals as they become unprofitable because of obvious udder disease, low milk yield, age or other reasons, and their replacement by younger animals takes, in the normal course, some years, and during all this time stringent precautions are necessary to avoid further spread. With the advent of modern specific treatments, progress became faster (Steck, 1938; Schalm, 1941, 1942 and 1943; Little, 1941, 1942; Martin, 1942; Kaestli, 1937, 1948) and when penicillin became readily available, with its almost complete freedom from irritant effects, which meant that infected carriers in full milk could be treated without fear of serious loss of yield, and its ability to work in pus-like secretions, and failure to give rise to resistant strains, progress seemed likely to be further accelerated. Our early experience with penicillin, even in herds which were under weekly test and in which treatment was immediate, was, however, like our earlier experience with the acridine dyes, in that whilst herd infection rates could be brought to a low level, eradication often eluded us. Meanwhile, it was shown by workers at other centres in Great Britain (Report 1944) and our own experience that *Str. agalactiae* became spread much more widely on the outside of the cow's udder and body and in her environment than had previously been recognised and that it could survive in these places for long periods.

Consideration of our results showed also that, whilst certain cows resisted all attempts to cure them, the majority responded at once and that a large part of the post-treatment infections detected at a given test was commonly in cows which had not previously been infected, *i.e.*, were new infections and not recrudescences; and this, despite the weekly herd tests, immediate treatment and sanitary precautions which were as good as are normally practicable. It seemed possible, therefore, that some of the difficulty in eradication of infection from many herds might be attributable to early infections undetected at a given test or to infection from intermediate sources, such as the external surface of the teats, milkers' hands, bedding, etc.

We had at this time an experimental herd in which 13 out of 20 cows had established infections in 27 quarters, in which there was more than an average amount of obvious mastitis (Lancaster & Stuart, 1949) and in which *Str. agalactiae* had been recovered with regularity from the teats of most of the cows, as well as commonly from other parts of the cow's skin, milkers' hands, overalls, utensils, udder cloths, bedding, shed fitments, and at times from walls, floors and even the air. This herd provided a severe test as to whether there was any advantage in simultaneous intensive treatment of all quarters of all cows infected or not, combined with thorough disinfection of cows and premises and it was decided to test this hypothesis. The result was immediate eradication (Stuart & Lancaster, 1949). The cows were again infected, this time by udder injection, and subsequently retreated. All infections were again sterilised except in one cow which was removed. The majority calved again and all were still negative. This result was so striking that it was decided to try the method in a severely infected farm herd and, if successful, as it proved to be, to test it in ten herds. The object of this report is to present the results obtained.

Procedure

METHODS OF MILK EXAMINATION

Samples of milk were taken with the usual precautions into 1 oz. screw-capped bottles. After inspection for abnormality, direct examination was made by spreading 0·1 ml. on the surface of a sheep blood agar plate without dye or aesculin, and reading after 48 hours at 37° C. The remainder of the sample was incubated overnight and a loopful of gravity cream spread on half of an Edwards plate (crystal-violet aesculin blood agar) and the results read after 24 hours' incubation, *i.e.*, at the same time as the direct plate. Colonies were identified by inspection and group precipitation tests with acid extracts (Lancefield) by the contact methods at room temperature (Stableforth, 1937), supplemented by biochemical tests when necessary as, *e.g.*, with *Str. uberis*. The kinds and numbers of each potential pathogen were recorded at each test.

In the earlier stages in the first herds quarter samples were examined. It was then decided that since all quarters were to be treated, cow samples should be used. Very little more difficulty is found in reading results, and this is repaid by the larger number of samples which can be tested.

Samples were examined twice before treatment, the second occasion being at the milking immediately before treatment, twice in the week following treatment, weekly for a month or until four weekly herd tests had been passed, then monthly for three months, and finally at about 6, 9, 12 and 24 months, short interval tests being resumed if infection was found.

EXAMINATION OF TEATS, SKIN, HANDS, UTENSILS, ETC.

Details of experiments with various methods for the examination of sites other than the milk are being described elsewhere (Chodkowski, 1949, in course of publication). The methods used for these ten herds were briefly as follows. Two media were used (*a*) a selective liquid enrichment medium consisting of sterile milk containing final concentrations of 1 : 20,000 potassium tellurite and 1 : 500 boric acid tubed in 10 ml. volumes; and (*b*) a selective solid medium consisting of sheep blood agar plates containing final concentrations of 1 : 750,000 crystal violet, 1 : 3,000 thallium sulphate, and 1 : 1,000 aesculin. Samples of each batch of medium were tested with known materials before use for herd examinations in order to check sterility and efficiency.

Sterile cotton wool swabs, moistened in sterile milk, were rubbed on the site to be examined, agitated in the liquid enrichment medium previously tubed in 10 ml. volumes, squeezed out against the side of the tube and discarded. The enrichment medium was incubated for two to three hours at 37° C. and 0·1 to 0·3 ml. was spread with a glass rod on the solid medium. After 48 hours the plates were examined and any *Str. agalactiae* colonies identified by their appearance and checked by serological and, in some cases, biochemical tests.

In the case of teats, one swab was used for all four teats before any washing had taken place. The skin swab included several sites, normally the flanks, outside of both hind legs and upper surface of the base of the tail. Swabs from clothes were usually from the front of the overalls, but at times from trousers, coats, caps and boots. "Floors, etc." include stall partitions; "cleansing utensils" include brooms, shovels, forks and the hose pipe; "milking utensils" include stools, the outside of hands and machine buckets and strip cups. The towel was that used for wiping hands. In all of these cases swabs were taken from the places most likely to be handled and infected with *Str. agalactiae*. "Other sites" include the inside of milking buckets, the milk cooler, dairy table, dairy brushes and sink, etc., places from which it would not be expected that cows would be directly contaminated. In general, examinations were made before milking was begun, and, in any case, swabs from a given animal were taken before she was handled.

"Hands" were examined after ordinary washing. They were then scrubbed with a sterile brush in 200 ml. sterile milk for three to five minutes and 10 ml. volumes of the milk were placed in tubes containing no dye, or one of several dye mixtures, described elsewhere (Chodkowski, 1949).

Air was examined by exposing 20 ml. volumes of sterile milk in open Petri dishes for five hours during the day or about 18 hours overnight at various places in the cowshed and then transferring the milk to a tube containing no dye or a mixture of dyes, to produce a liquid selective medium which had the same composition as the liquid medium used for testing the hands, and was later plated on the solid selective medium described above. More positives were obtained during the five-hour-day period than during the night. One examination was made of each herd before treatment and three afterwards, except in herd 8, where only two were made, and herd 10, where none was made. The pre-treatment test was made at or shortly before the date when treatment was begun. The first post-treatment tests were made usually at one to two months after treatment, but in three herds at three to six months and the last at four to ten months after, the delay in some herds being because examination of materials or sites other than milk was not introduced until some months after the first herd was treated.

Treatment

After the afternoon milking on five successive days, 100,000 units of penicillin were injected into each quarter of every cow, whether infected or not, in milk or dry. In the first seven herds the penicillin was dissolved in 10 ml. distilled water, a freshly boiled glass syringe and teat needle being used for each cow. In the other three herds, half the cows were treated in the same way. In the remaining cows in these herds, the penicillin was injected in 6 c.c. of 4·5 per cent. beeswax in arachis oil from *pure* tin tubes which had a fixed extruded injection nozzle protected by a plastic screw cap, sealed with paraffin wax.*

This method of injection was introduced to minimise the possibility of transmission of tubercle bacilli from cow to cow or herd to herd, following reports (MacFarlane and others, 1944; Ritchie, 1946) that it was a serious danger attendant on udder injections unless the use of sterile instruments always could be ensured. Tubes of this or a similar nature, but containing a smaller volume which facilitates injection, have been used in all subsequent work. The use of wax-oil suspension in tubes has also the advantages that the penicillin keeps well and is always ready for immediate use. Whilst stocks will normally be kept in the cold, loss of potency is very slow at ordinary temperatures. One of our colleagues (Dr. Slavin) has tested tubes at intervals and found no loss of potency up to six months' storage at laboratory temperatures. After 20 months they had lost about one-third. Administration of penicillin in oil-wax has another advantage, viz., that concentrations in the milk 24 and 48 hours after administration are more regular than after aqueous solutions.

Infections which appeared after the herd treatment were treated at once but no further mass treatment was done except in herd 12. In-calf heifers were not treated, but were sampled as soon as the colostral stage had passed and, where possible, before they entered the milking line.

Disinfection of Cows and Premises

Preliminary cleaning-up of the cows and premises was done by the farm staff during the early part of the week of herd treatment. On the morning of the fifth day each cow was in turn sprayed against the hair with a hypochlorite solution containing 1,600 p.p.m. Cl. delivered by a 20-gallon hand pump, and was turned out after spraying. Bedding and remnants of food and any removable objects were taken out of the byre or byres and these, together with calving boxes, yards and any other buildings used by the cows, were soaked with an officially approved cresol disinfectant and utensils and other equipment were appropriately sterilised. The cows were brought back at the afternoon milking when clean milking clothes and sterilised udder cloths and milking equipment were provided.

Penicillin cream, 1,000 units per gramme, was used on the milkers' hands and the teats whenever the cow's udders were handled, for 14 days from the first day of treatment.

Each farm was also kept supplied with a small pot of penicillin cream for use on heifers or any other animals added to the herd, until they had been tested. Pots withdrawn from farms after they had been lying in the cowshed for three to six months still showed about half the original penicillin content despite, in some cases, the presence of obvious dirt.

* We are much indebted to Messrs. May & Baker, Ltd., Dagenham, for their help in supplying penicillin in this form.

General Information *re* Herds

Herd 8.—Pedigree (Shorthorn) herd, average 50 milking animals, attested, vaccinated with S.19 vaccine, milked with a Gascoigne bucket plant at 15 lb. vacuum, hand-stripped, calves left on dam for four days in calving boxes and then moved to another farm where bucket fed, weaned, reared and served, being brought back when due to calve. Premises old, but well kept. Milking hygiene good. Hypochlorite used.

Herd 9.—Non-pedigree Shorthorn herd of about 40 animals vaccinated S.19 vaccine, hand milked, calves on dam for three days, when work begun but now removed at once and bucket fed on mother's milk for a few days, then on general supply, calves tied up for first few days or longer, but in groups by end of a month, and do not commonly suckle one another. Premises old, hygiene moderate, hypochlorite used.

Herd 10.—A herd of about 36 milking animals, mostly Friesians, but with some Shorthorns, not vaccinated against abortion, milked with an Alfa Laval bucket plant at 13 lb. vacuum, hand-stripped until recently, calves removed from dam at 24 hours and reared away, being brought back when due to calve. Premises good and milking hygiene excellent, hypochlorite used.

Herd 11.—Mixed herd of pedigree Red Polls and non-pedigree Guernseys of about 50 milking animals, vaccinated S.19 vaccine, T.T., milked in a Gascoigne auto-recorder combine bail at 13 lb. vacuum, kept in yards in winter, hand stripped, calves left on dam for four days, some then put on suckling cows, some bucket fed, not more than two calves kept together. Premises excellent and milking hygiene good, hypochlorites used.

Herd 12.—Herd of about 20 milking Guernseys, being graded up, and many pedigree. Attested, vaccinated S.19 vaccine, milked with a Gascoigne bucket plant at 13 lb. vacuum, general hand stripping ceased during period under control and now only a few of the older cows stripped, calves left on dam for three to four days, then on nurse cows which take several calves; calves do not commonly suckle each other, but can. Milking hygiene good, hypochlorite used for first year of work but teat sores a problem and now no disinfectant used. Premises good and well kept.

Herd 13.—Pedigree Friesian herd of about 30 milking head with high yield, undesignated but now becoming T.T., vaccinated S.19, milked with a Gascoigne bucket machine when work began, now with a Gascoigne combine auto recorder at 15 lb. vacuum, hand stripped, calves suckled once on dam or overnight, then bucket-fed and kept singly. Milking hygiene good, hypochlorite used. Premises moderate and reasonably well kept.

Herd 14.—Non-pedigree Shorthorn herd of about 30, not vaccinated originally but now with S.19, milked with an Alfa Laval bucket plant at 13 lb. vacuum, hand-stripped when work began but not during last 18 months, calves left on dam for four days, then bucket-fed and kept separate for three months. Cowshed poor and all premises old, milking hygiene moderate.

Herd 15.—A mixed herd of Friesians and Jerseys in which we ceased observations after 12 weeks because staff was totally inadequate, hygiene poor, fresh cows appeared at almost each test, and co-operation was not practicable.

Herd 16.—Pedigree Friesian herd of about 20 cows, vaccinated S.19, milked with a Gascoigne bucket plant at 13½ lb. vacuum, not hand-stripped, calves removed from dam at once, bucket fed, and kept in twos, some suckling each other. Cowshed good and milking hygiene excellent.

Herd 17.—Herd of about 30 milking head, non-pedigree Shorthorns when work began, now about half are pedigree Guernseys, vaccinated S.19, milked with a Gascoigne bucket plant at 15 lb. vacuum, hand-stripped, calves were originally left on dam for three days, but now removed at once and bucket-fed. Premises and hygiene moderate, hypochlorite used.

Results

Str. agalactiae Infections Before and After Herd Treatment

In Table I are shown the number of milking animals in each herd, in milk or dry, before the herd treatment was done, the numbers infected with *Str. agalactiae* before treatment, and the numbers found infected in each month subsequently. The pre-treatment figures are the total found at the two tests before treatment. Most animals were positive at both tests and showed infection in the direct examination on plates. A few were positive at one test only or only to the enrichment test. The figures for the first month post-treatment and for any month following an infection are the total results of weekly tests. Other figures are in general those of a single monthly or longer period test. All positive results, whether by direct or enrichment test, are included.

TABLE I

TOTAL *Str. agalactiae* INFECTIONS IN TEN SUCCESSIVE HERDS BEFORE AND AFTER TREATMENT OF ALL QUARTERS OF ALL COWS WITH FIVE INJECTIONS OF 100,000 UNITS PENICILLIN AT DAILY INTERVALS AND DISINFECTION OF COWS AND PREMISES

Herd	Size	Numbers infected before and after herd treatment: Before	After (in months) 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
8	50	25	1	0	0		0		0		0			0						0						0
9	39	17	9	2	0	1	0	0	0			0				2		0							0	
10	36	17	0	0	0	0	0		0			0	0			0	Herd sold									
11	48	2	0	0	0	0				0	0			0												
12	19	5	0	1	1	2	0	0	0*	2	10	Herd treated again			0	0	6		0			1	0			
13	30	19	1	4	0	0	1	1	0	0	0			0												
14	30	10	0	0	0	0	0		0	0		0				0								0		
15	119	22	3	2	17	Work stopped; lack of farm staff and co-operation																				
16	21	7	0	0	0	0		0				0				0							0			
17	32	24	0	0	0	0		0		•	5	3	0	0	1	5	3	5	7	2	5	1				
	424	148 (35%)														+6	+16	+1	+1	+2	+1					

* In these herds an infected cow was introduced in the 8th month.
(At the original herd treatment all cows were treated. Thereafter, only newly infected cows (if any), were treated except in herd 12 in which a second herd treatment and disinfection was done.)

It will be seen that of 424 cows in these ten herds, 148 (35 per cent.) were infected with *Str. agalactiae*, and that the herd incidence was from 26 to 75 per cent., except in herd 15, in which work was stopped at the twelfth week, and herd 11, which was included because of the high incidence of other infections.

Of these 148 infected cows, 94 per cent. were negative to direct cultural tests up to two months after treatment, and 90 per cent. to enrichment tests. The larger number of infections in Table I includes recrudescences after retreatments and new injections in previously negative cows.

In herd 8 one cow, treated when dry, was again found infected at the third weekly test but responded to a second course of treatment. Since then the herd has remained free up to the test at the end of two years.

In herd 9 many recrudescences of infection, or new infections occurred during the first two months and until a cow which had failed several treatments was removed to the laboratory. It was found by special methods of sampling that the infection detected in her samples was not due to infection of the inside of the udder but to infection in a sore near the tip of the teat and when this was cured milk samples became negative. Infection continued to be present on the outside of the teats of a variable but large percentage of cows for many months after the herd treatment (see Table II and comment thereon), and infection was found in milk samples from two cows in the 14th month. From then until the last test at 23 months none was found and the herd now appears to be free.

In herd 10 treatment was at once successful in all cows and no further infection was found up to the last herd test at 14 months. Samples from newly calved animals after that time continued to be negative and two animals purchased by the laboratory at the sale were negative.

In herd 11, which had only two infected animals, eradication was also achieved at once. Samples received since the last herd test at 12 months have also been negative.

In herd 12 heavy infection persisted on the outside of many udders for many months (see Table II) and milk samples were also found infected in the second, third and fourth months. During the next three months no infection was detected in milk samples, but infection continued to be found on the outside of many teats, and by no means always in the same cows. It fell to 0 to 10 per cent. by the sixth month, remaining at that level until 7½ months when it rose again. During the eighth month a herd examination of milk samples showed infection in a cow which had previously been free and also in one which had been introduced and during the next month infection spread widely, the teat carrier rate also reaching a high level. Treatment of individual infected animals was stopped until the whole herd could be retreated. This was done in the 13th month and all herd tests, weekly and then monthly, were passed until the 20th month, when one infection was again found. This was, as usual, treated at once and no further infection has been detected up to the date of report, one month later.

Herd 13 showed five infections in the first two months post-treatment, of which two were in newly-calved heifers and one infection in each of the fifth and sixth months. It then remained free up to the herd test at 12 months, whilst all samples since received have been negative.

In herd 14, *Str. agalactiae* was eradicated at once, the last herd test being 22 months after treatment, calving samples since received being negative.

In herd 15 infections were found in each month after treatment and reached a high level again by the third month, when work was stopped for reasons already given.

In herd 16 eradication was achieved at once, the last herd test being at 21 months, calving samples having since been negative.

In herd 17 it is also believed that all infection was eradicated by the herd treatment and disinfection because no infection appeared thereafter up to and including the six months' milk test or at any of the three post-treatment examinations of other sites (see Table III). At the herd test at nine months, however, infection was found in a cow which had been purchased a month before and added to the herd without test, and four other cows were detected at weekly tests in the same month. Control by immediate treatment of infected cows and general tightening-up of hygiene seemed to be within reach by the twelfth month, but infection has since spread widely and so far defeated all attempts to bring it under control again. The number of infected milk samples became so high in recent months that treatment of all new infections became uneconomic and only those carrying heavy infection have been treated; the others, which are shown as a + figure (5 + 6, etc.) in Table I, showed a few *Str. agalactiae* and were left untreated to see what would happen; many proved negative at the next test. At the same time certain cows which resisted several treatments, or appeared to be the main sources of infection, have been removed and events have recently taken a better turn. This herd, however, exemplifies the difficulties which may be met with in certain herds. The new infection may be with a strain of specially invasive properties but only further work can shed light on this. No clinical mastitis has yet occurred nor any clotting of milk or reduction of yield.

To summarise, work was suspended early in one herd because of lack of farm co-operation. Six herds (Nos. 8, 10, 11, 14, 16 and 17) were freed at once, except for one cow in herd 8 which failed to the herd treatment but responded at once to retreatment, and five of these herds have remained free for 12 to 24 months, whilst one was reinfected by a purchased cow. One herd (No. 13) showed some infection up to six months but has since remained clear. One herd (No. 9), after some early trouble and since the finding of two infections at 14 months, has now remained clear for eleven months. One herd (No. 12) which had a new outbreak in the eighth month due either to an infected cow which was introduced or to infection which had not entirely disappeared from the outside of the teat, was retreated and has shown one infection since.

Streptococcus agalactiae IN SITES OTHER THAN MILK

The results summarised in Table II show that *Streptococcus agalactiae* could be cultivated before milking from a high percentage of the teats, from the hands of over one-third of the milkers after the normal washing, and at a considerable proportion of tests of other places handled by milkers or with which the cows themselves came into contact.

They also show that in five herds it could not be obtained anywhere at a large number of examinations made after treatment and disinfection and that in a sixth herd (No. 16) in which all cows

were freed by the herd treatment and remained free, it was obtained once only, viz., from a swab from a floor two months after disinfection. In another herd (No. 14), in which also the milk of all cows remained free from the date of herd treatment, it was found on the teats of five cows and on the udder cloth, five months after the herd treatment and not at the many tests made before or after. These two latter findings are puzzling. In herds 9 and 12, on the other hand, infection persisted for the three examinations covered in Table III, and also, despite the use of various creams and solutions before or after milking, for a much longer period. In herd 9, which is now free, the source may have been through a milker who also at times milked his father's cows, which we could not examine. In herd 12 teat sores were a serious problem and were frequently heavily infected and they may have been the source of the persistence. The sores eventually decreased to a low level and the herd was treated and disinfected again. It is not known whether the persistence of this teat infection played an important part in the new outbreak in herd 12 or whether the infected cow which was introduced was mainly responsible.

Infections with *Str. dysgalactiae*, *Str. uberis* and Haemolytic Staphylococci

In Table III are shown for each herd the average number of cows infected with the above organisms before and after treatment. Averages are used because, whilst chronic carriers of *Str. dysgalactiae* and haemolytic staphylococci and, more often, *Str. uberis* occur, a large number of infections occur once, or maybe twice, in the same cow, but all other tests are negative; in other words, whilst enduring infections do occur, the majority appear to be sporadic, the cow either overcoming the infection, which may never gain a hold on the tissues completely, and without symptoms or after a subacute or acute mastitis. This contrasts with the usual course of *Str. agalactiae*. The figures for before treatment are the average of two herd tests. The post-treatment figures are the average of two to four tests where short interval tests were being made, *i.e.*, in the early stages in each herd or until it was believed *Str. agalactiae* had been eradicated; otherwise they are usually those of single tests.

It will be seen that whilst rises and falls in the incidence of one or other organisms occur and a marked rise and peak covering many months occurred at one period in herd 12, there is no general evidence of any increase during the period since herd treatment was given, and that, in fact, the incidence of all these other organisms usually decreased following herd treatment and has generally remained at lower levels than previously.

The general findings regarding the efficiency of the herd treatment against various organisms was that *Str. dysgalactiae* infection was usually sterilised, and that the response of *Str. uberis* was variable, whilst staphylococcus infection, though usually reduced, was not usually eliminated.

Clinical Results

In Table IV are summarised on the left side the mastitis history as far as it could be elicited and the clinical findings on the day of the first visit to each herd, and on the right side the number and nature of the clinical cases which have occurred in the 20 to 25 months which have elapsed since the herd treatment was carried out.

A detailed clinical history was not always possible, but it will be clear that mastitis was a serious problem in most of the herds. The records of the abnormalities at the time of the first visit are

Table II

Str. agalactiae in Sites other than Milk Before and After Herd Treatment; Proportions and Percentages in Nine of the Ten Herds

Herd	Before herd treatment and disinfection												
	Cows		Milkers, cowsheds, equipment and dairy										
	Teats	Hair or skin	Hands	Clothes	Floors, etc.	Cleaning utensils	Udder cloths	Machine cups	Milking utensils	Towels	Churns	Air	Other sites
8	6/25(24)	—	0/2	0/1	0/4	1/2	—	—	0/1	—	—	—	1/2
9	50/77(65)	6/77(8)	4/5	—	0/4	2/3	—	—	1/2	—	0/1	4/4	1/2
10	7/27(26)	2/33(6)	2/2	—	1/4	0/4	—	—	—	—	—	0/3	—
11	5/48(10)	0/28	0/2	0/1	0/1	0/3	—	—	0/1	—	—	0/2	0/3
12	5/19(26)	0/19	0/2	0/2	0/6	0/1	—	—	—	—	—	0/5	0/2
13	12/3 (40)	0/30	1/3	0/2	0/2	0/2	—	—	2/4	—	—	0/3	1/4
14	4/28(14)	0/28	1/3	—	0/7	0/2	—	—	1/1	—	—	0/5	2/6
16	17/26(81)	2/21(10)	1/2	1/6	2/14	1/10	—	0/3	0/1	—	—	0/3	2/8
17	27/32(88)	2/32(6)	0/2	1/6	3/15	0/9	3/3	0/2	1/1	0/1	—	0/2	2/9
Totals	133/3 7(43)	12/268(4)	9/23	2/18	6/37	4/36	3/3	0/5	5/11	0/1	0/1	4/27	9/36

Herd	After herd treatment and disinfection													
	Cows		Milkers, cowsheds, equipment and dairy											
	Teats	Skin	Hands	Clothes	Floors, etc.	Cleaning utensils	Udder cloths	Machine cups	Milking utensils	Towels	Churns	Air	Other sites	*Str. agalactiae* present or absent
8	0/73	0/43	0/5	0/9	0/12	0/3	0/4	—	0/2	—	0/1	—	0/7	Absent
9	1/42(2)	1/42(2)	0/3	—	0/6	0/1	—	—	0/1	—	—	0/3	—	Still present
	19/37(51)	2/37(5)	3/4	2/3	0/12	1/5	1/2	—	0/3	—	0/2	0/3	0/1	" "
	22/37(60)	—	2/4	2/8	—	—	1/1	—	—	—	—	0/3	—	" "
10	0/105	0/71	0/6	0/7	0/25	0/7	0/5	0/4	0/8	0/2	0/1	0/7	0/9	Absent
11	0/130	0/94	0/9	0/4	0/6	0/1	0/5	—	0/5	0/1	—	0/2	0/5	Absent
12	4/19(21)	0/19	0/2	0/4	0/7	0/5	0/3	—	0/1	—	—	0/2	0/5	Still present
	12/19(63)	—	—	—	—	—	—	—	—	—	—	—	—	" "
	9/19(46)	—	—	1/2	—	—	0/1	—	—	0/1	—	—	—	" "
13	0/87	0/29	0/3	0/18	0/8	0/4	0/4	—	0/6	0/2	0/4	0/3	0/13	Absent
14	0/24	0/24	0/1	0/2	0/8	0/2	0/1	0/1	0/3	0/1	0/1	0/3	0/8	Absent
	5/23(22)	—	0/2	0/1	0/5	0/2	1/1	—	—	0/1	0/5	—	—	Present
	0/24	—	0/2	0/1	0/3	0/1	0/2	—	0/1	—	0/1	0/1	—	Absent
16	0/163	0/42	0/6	0/5	1/23	0/5	0/3	0/2	0/4	0/2	0/3	0/7	0/16	Absent
17	0/98	0/66	0/5	0/6	0/17	0/6	0/16	—	0/5	0/2	0/3	0/6	0/7	Absent

Note.—Where the results of all examinations after treatment were negative they are added together and shown in one line (herds 8, 10, 11, 13, 16 and 17).

those of animals with definite udder changes or, in most cases, abnormal milk. The after-treatment data almost certainly include all, or nearly all, cows which showed abnormality because it was a standing request that samples should be sent in from such animals and the laboratory was supplying penicillin free of charge and also defraying the costs of veterinary attention, which the owner called direct in acute cases on the understanding that a sample was sent. The data for post-treatment cases, therefore, include minor cases such as would not have been remembered or recorded in the pre-treatment histories. Despite this, it will be seen that only 51 cases were recorded in 304 cows over a period averaging 22 months, or less than one per eleven cows per year, and that in only three cases in all was a quarter lost, one of which was due to *C. pyogenes* and one to *Cl. welchii*. Most of the cases were mild or subacute and responded at once to treatment. They have been distributed fairly evenly over the period. The owners of all herds except No. 11 are more than pleased with the results, not only because of the low incidence of udder trouble of any kind, but because the occurrence of flakes or clots on the discs or cloths used for filtering or straining the milk is exceptional, except when a new calved or nearly dry animal is in the herd and then, apparently, much less common than previously. Herd 11 has shown much more clinical mastitis since herd treatment than the other herds. Only one case was serious but the difference in the position before and after herd treatment is much less striking than in the other herds, presumably because most of the previous mastitis was due to organisms other than *Str. agalactiae*, *cf.* Table I. It is worthy of note that even in herds 2 and 13, where eradication was delayed, and in herd 17, where it was reintroduced and is not yet under control, clinical *Str. agalactiae* infections have been rare, whilst in the other herd, which was reinfected with considerable consequent spread, none occurred. *Str. dysgalactiae*, *Str. uberis* and haemolytic staphylococcus cases are certainly not more, and probably much less, than would be expected in untreated herds.

TABLE III

AVERAGE NUMBER OF COWS INFECTED WITH *Str. dysgalactiae*, *Str. uberis* AND *Haemolytic staphylococci* IN TEN HERDS, BEFORE AND AFTER TREATMENT OF ALL QUARTERS OF ALL COWS WITH FIVE INJECTIONS OF 100,000 UNITS PENICILLIN AT DAILY INTERVALS AND DISINFECTION OF COWS AND PREMISES

Herd	Number of cows	Bacterium	Numbers infected before and after treatment																							
			Before	After (in months)																						
				1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
8	50	*Str. dys.*	1	0	0	1		0		2		1			1						2						3
		„ *uberis*	2	2	4	3		4		1		0			2						1						3
		H. staph.	7	6	5	4		1		0		2			1						0						0
9	39	*Str. dys.*	0	0	0	0	0	0	0	0			0				0		0							0	
		„ *uberis*	0	0	1	1	1	2	2	1			3				2		1							3	
		H. staph.	6	2	2	3	2	4	3	1			3				0		0							0	
10	36	*Str. dys.*	1	0	0	0	0	2		1			0	0			0										
		„ *uberis*	0	0	1	1	2	2		1			0	1			0		Herd sold								
		H. staph.	2	1	1	2	1	0		1			2	0			0										
11	48	*Str. dys.*	4	0	0	0	0				0	1			0												
		„ *uberis*	8	0	0	0	1				1	2			1												
		H. staph.	12	1	1	1	2				0	1			1												
12	19	*Str. dys.*	0	0	0	1	0	0	0	0	0	0	Herd			0	0	0		*1			3	3			
		„ *uberis*	3	1	0	1	0	0	0	0	1	2	treated			0	1	0		1			1	1			
		H. staph.	2	1	3	4	7	3	2	4	2	2	again			1	1	1		0			0	0			
13	30	*Str. dys.*	0	0	0	0	0	0	0	0	1	0			1												
		„ *uberis*	1	0	0	0	0	0	0	0	0	0			1												
		H. staph.	4	1	1	2	4	1	1	0	0	0			1												
14	30	*Str. dys.*	0	0	0	1	0	0		1	0		0				0										
		„ *uberis*	0	0	1	0	0	0		0	1		0				1										
		H. staph.	5	1	2	2	3	1		1	1		1				1										
15	119	*Str. dys.*	1	0	2	1																					
		„ *uberis*	5	0	1	2	Work discontinued																				
		H. staph.	5	1	2	7																					
16	21	*Str. dys.*	0	0	0	0	0		0				0				0										
		„ *uberis*	1	0	0	0	0		1				0				0										
		H. staph.	2	1	0	4	1		2				0				0										
17	32	*Str. dys.*	4	1	0	2	0		0		0	0	0	0	0	0	0	0	0	0	0	0	1	1			
		„ *uberis*	0	0	2	1	1		0		2	0	0	0	1	1	1	1	1	1	1	1	1				
		H. staph.	0	1	1	1	0		0		0	1	0	1	0	0	0	, 0	0	0	0	0	0				

TABLE IV

CLINICAL MASTITIS BEFORE AND AFTER HERD TREATMENT

Herd	Size	Mastitis history before herd treatment	Clinical cases after treatment							
			Period in months	*Str. agalactiae*	*Str. dysgalactiae*	*Str. uberis*	*Haem. staphylococci*	Others	Total	Quarters lost
8	50	Typical history of recurrent trouble, 12 quarters of 9 cows obviously abnormal at first visit	25	0	6	1	1	1	9	0
9	39	As herd 8, 12 cows abnormal at first visit	23	2	1	1	0	3	7	0
10	36	Much trouble in previous year, 5 serious cases in previous 4 months, 5 cows abnormal at first visit	21	0	0	1	1	1	3	0
11	48	Little chronic mastitis but many acute or subacute cases, 10 cows abnormal at first test	22	0	6	4	2	4	16	0
12	19	Clots from time to time ; 9 cows abnormal at first visit	22	0	0	0	0	0	0	0
13	30	Serious, 21 cows removed in previous 2 years for mastitis ; 4 abnormal at first test	22	1	1	1	0	1	4	1
14	30	Serious, 5 cows recently removed and 9 affected at first visit, 4 severely	22	0	0	1	0	2	3	1
16	21	Serious, 13 cows treated in previous 6 months, 4 abnormal at first visit	21	0	0	0	5	1	5	1
17	32	Serious, 11 cows abnormal at first visit	20	1	0	2	0	1	4	0
Totals	304	Over an average of	22	4	14	11	9	14	51	3

Effect on Milk Yields

Some reduction in milk yield occurred on most farms during the week of treatment and disinfection but, except on two farms, it was less than 5 per cent., *i.e.*, not more than would be accounted for by the presence of strangers and disturbance of routine. On the two exceptional farms, which were treated in successive weeks with aqueous solutions of penicillin, greasy slimy clots occurred in a large proportion of cows on the morning after the second injection. They persisted until next day and then gradually disappeared, although treatment was continued, and were practically gone by the fifth injection. It is not certain whether this was connected with the particular batch of penicillin used but, except in rare individual cows, nothing of the kind occurred in any of the other herds, nor has it occurred in any of over 50 herds since treated.

In most of the herds, milk yields have increased very considerably since herd treatment, both in respect of the daily average per cow in milk and in respect of yearly yields and increases of 50 to 200 gallons per cow are not uncommon. Often an increase occurred soon after treatment, but in many cases it was more evident in the next lactation. It is the general opinion of owners that this is due to the great reduction or virtual elimination of mastitis following treatment. Other factors, however, may be concerned, and it is intended to leave this aspect of the work until all previously infected cows have completed the lactation subsequent to the one in which they were treated, when an analysis will be made with corrections for all other factors which might be concerned.

Summary

1. Attempts have been made to eradicate *Str. agalactiae* from ten herds by simultaneous treatment of all quarters of all cows with five doses of 100,000 units penicillin at daily intervals, disinfection of cows and premises, and use of penicillin cream on udder and hands for 14 days after the beginning of herd treatment and for any cows subsequently infected or added to the herd.

2. The herds contained 424 cows, of which 148 (35 per cent.) were infected with *Str. agalactiae* and eleven (3 per cent.), 20 (5 per cent.), and 45 (11 per cent.) showed *Str. dysgalactiae*, *Str. uberis* and haemolytic staphylococci respectively before treatment (average of two tests).

3. *Str. agalactiae* was eradicated from five herds at once, from another herd within three weeks by retreatment of one cow, from another in six months by subsequent treatment of seven cows (two being heifers infected at first calving), and from another herd in 14 months by subsequent treatment of 14 cows. Many of these treatments were retreatments of the same cows. In another herd, a second herd treatment was made; it is free at present but the eventual outcome is not yet known. An infected cow was introduced into this herd in the eighth month and also into one of the herds which were freed at once, and in both cases considerable spread occurred. Seven herds are now free. Work was stopped early in one herd because of lack of farm co-operation.

4. By pre-treatment examinations made on selective media before milking, *Str. agalactiae* was obtained from the outside of the teats of 43 per cent. of cows, from the skin of 4 per cent., from 39 per cent. of milkers' hands after the ordinary washing, and from a variety of other sites from which cows might be infected directly or through hands. After treatment and disinfection, *Str. agalactiae* was absent in five herds, was detected in two of the cleared herds on one occasion under circumstances suggesting an outside source, and persisted on a high percentage of teats and, in certain other places, in two herds in which infection also occurred again in milk samples.

5. *Str. dysgalactiae*, *Str. uberis* and haemolytic staphylococcus infections were not eradicated but were reduced and have not reached pre-treatment levels, nor caused more trouble than earlier.

6. Clinical mastitis has ceased to be a problem in all nine herds in which observations were continued. A total of 51 cases, including those which showed only a few clots, and most of which were mild, occurred in the 304 cows in the nine herds over the period of 20 to 25 months (average 22 months) or one per year per every eleven cows. Only three quarters were lost.

7. Milk yields have increased considerably and an analysis will be reported at a later date.

8. In the earlier treated herds the penicillin was injected in 10 ml. of distilled water. In three herds it was injected from pure tin tubes fitted with a fixed extruded teat nozzle and a screw cap sealed with paraffin wax, which delivered the 100,000 units penicillin in 6 ml. of 4·5 per cent. beeswax in arachis oil and which were discarded after use. Use of this method minimises the possibility of transmission of tubercle bacilli or other penicillin-resistant bacteria from cow to cow or herd to herd. It has also the advantages that the penicillin keeps well, is ready for immediate use, and that concentrations maintained in milk appear to be more regular than with aqueous solutions.

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ROYAL VETERINARY COLLEGE ATHLETIC CLUB

The Royal Veterinary College Athletic Club held their third annual sports day on May 28th on the Goring Recreation Field, Goring.

On this occasion the programme took the form of an inter-year competition, the winning year—Final Year—receiving the Greatorex Challenge Cup.

The most interesting event proved to be the tug-o'-war, teams here competing for the Tanner Trophy. Much amusement was caused when the winning tug team (Third Year) were challenged by Members of Staff to a "pull." Fortunately Third Year proved a trifle too strong.

Established College records mainly stood the test, although one was broken—Long Jump, by M. Muse (Final Year). Ladies' events were introduced for the first time and competition was very keen. The mixed relay also proved most successful.

The Principal, Professor J. B. Buxton, was present and addressed the students at the presentation of medals and trophies, expressing his pleasure at having attended such a delightful Sports Day.

Mrs. Buxton, who kindly presented the medals and trophies, was presented with a bouquet by Miss Jennifer Greatorex. Mrs. Greatorex was also presented with a bouquet in recognition of her help to the committee.

The day proved an unqualified success, being concluded in the evening by a Carnival Dance held in the Morrell Rooms, Streatley.

Winners of events were as follows:—

100 Yards, Muse, M.; 220 Yards, Smith, E. H.; 440 Yards, French, P.; 880 Yards, French, P.; One Mile, Boys-Stones, P.; Three Miles, Watson, K.; 80 Yards (Women), Miss Ibbetson, Miss Walker (*dead heat*); Discus, Webster, A.; Javelin, Briault, M.; High Jump (Men), Keep, M. E.; Long Jump (Women), Sutherland, A.; Long Jump (Men), Muse, M.; High Jump (Women), Walker, G.; Shot, Gillett, T.; Hop, Step, Jump, Tanner, S. C.; Medley (Relay), Third Year; Mixed Relay, Fifth Year; Tug-o'-War, Third Year.

Final Result.—Fifth Year, 74½ points; Third Year, 35; First Year, 34; Fourth Year, 23½, and Second Year, 19.

So-Called "Hard Pad" Disease*

By

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SEVENOAKS

Last August, MacIntyre, Trevan and Montgomerie presented a paper entitled "Observations on Canine Encephalitis" at the Southport Conference, and this was published recently in the *Record*[1], together with the observations of the various contributors to the discussion. There was some disappointment on that occasion, due to the fact that so little time was available for the subsequent discussion. In these circumstances, and especially in view of the fact that you will have had ample opportunity to read their paper before coming here to-day, I propose to give only a brief résumé of the main clinical characteristics of this syndrome as I have encountered it in the dog, in order that plenty of time will be left for a full discussion by the many practitioners present to-day. Some of the deductions I have been tempted to make may be rather provocative, but this is not unusual in so controversial a subject. After all, there seem to be at least five schools of thought on the nature of this infection:

(1) Those who believe H.P. (hard pad) to be a completely new disease.

(2) Those who believe H.P. to be due to L. and D. (Laidlaw and Dunkin) virus in a neurotropic form.

(3) Those who look upon H.P. virus as an accidental or aberrant form of the L. and D. virus.

(4) Those who think that H.P. virus has been with us for a long time and has frequently been mixed up with or mistaken for L. and D. virus.

(5) Those who consider the condition to be due to Beta haemolytic streptococcal infection complicated by bowel parasites (*vide The Times*, January 19th, 1949).

Introduction

I think it is true to say that most of us, after the publication of Laidlaw and Dunkin's[2] work in 1926, were confident that all the varying so-called forms of canine distemper (the pneumonic, the gastro-enteric, the nervous, etc.) would be controlled by the immunising methods devised by those workers, provided, of course, that adequate care was taken in carrying out their recommendations. Unfortunately, as time went on, illnesses following inoculations and "breakdowns" in immunised dogs occurred and at times shook our confidence greatly. We tried to explain away these occurrences varyingly, ascribing them to faulty technique, defective material or to a flare-up of a latent infection. There was, however, one thing that seemed common to all these setbacks and it was the fact that nervous phenomena almost invariably dominated the picture. Now, although these happenings had been going on since the publication of L. and D.'s work, circumstances were such that a thorough full-scale investigation was not carried through and published till after the war. It is no disrespect to the very excellent work of MacIntyre, Trevan and Montgomerie when I say that this canine syndrome flared up in rather a distinctive and persistent form during the period 1945–48 and permitted the investigators to obtain masses of invaluable clinical and pathological material on which to work. I used the word "distinctive" purposely, because this outbreak has kept remarkably clear of secondary septic invaders such as gave rise to purulent eye discharges, corneal ulceration, "brown mouth," middle ears, etc., in some former outbreaks—complications which greatly increased one's difficulties in sorting out this infection from L. and D. infection. I think it is possible that the widespread use of sulphonamides and penicillin has contributed to this end. In spite of this, however, H.P. disease is curiously protean in character, seeming to vary considerably in different parts of the country. Last summer, I. MacIntyre reported a majority of cases as developing severe pneumonia in the Edinburgh district, whilst nerve complications were comparatively rare, whereas W. L. Weipers, in Glasgow, reported a good admixture of the two. In the same period, in West Kent, the pneumonic form was uncommon but the nervous form was frequent and very severe.

Dogs of all ages are affected, but young dogs are certainly more liable to a fatal termination. The ages of dogs in an infected kennel will sometimes contribute to the development of the disease in several forms, quite apart from the nervous phenomena which are naturally most varied.

Symptomatology

H.P. disease is usually ushered in by fever, tonsillitis and mild adenitis. The primary rise of temperature, which is frequently high (105° to 106° F.), shows a tendency to fall back rapidly to 102° to 102·5° F. within 24 hours. On this account the primary rise is often missed. The subsequent fever is curiously low, often only hovering just above normal with an occasional rise to 103° F. It does not show the diphasic character of L. and D. infection.

There is invariably an early development of a diffuse injection of the sclerotic, with little tendency, however, to the development of a purulent discharge. There is, instead, rather a characteristic moisture around the eyelids, combined with a lack of lustre in the eyes themselves. Absence of this eye change in a dog that has been suspected of H.P. for several days would, in my opinion, eliminate this disease as a possibility. Conversely, the persistence of this eye change, even several months after apparent recovery, should warn one of the possibility of the development of encephalomyelitis under some stress such as excessive fatigue, oestrus in the bitch, inoculation of L. and D. virus, etc. This tardiness of some dogs in the field to develop a really active immunity would appear to be rather characteristic and such animals might well behave as "carriers" in this interim period.

There is not infrequently a thin mucoid nasal discharge which may later become slightly purulent and adhere as a dry crust to the edges of the nostrils.

During the first week there is no obvious pain when handling the pads but, at the end of this time, affected dogs may exhibit a certain amount of resentment at this procedure, and I have seen a few dogs walking in a manner similar to that of a laminitic pony. In the majority of cases, once actual hardness of the pads is easily palpable, it is only a question of time before symptoms of encephalitis develop. Unfortunately, the keratosis in many cases is not sufficient to give rise to any obvious changes in the pads and so put one on one's guard. One should mention at this point that in a large outbreak there are certainly a good number of non-clinical cases comparable to the dog that simply puts up a thermal reaction to inoculation of H.P. virus.

In spite of many reports of severe pneumonia elsewhere in the country, the West Kent area pneumonia, when encountered, has been relatively mild and responded well to penicillin. On the other hand, I have seen several severe and fatal cases of myocarditis.

Diarrhoea is a frequent complication and often proves most intractable to treatment, whilst the more severe cases develop dysentery. In those animals that apparently have recovered, there is a tendency to intermittent looseness with admixture of mucus in the stools. The possibility that such dogs may spread infection after recovery is one that should always be borne in mind, especially if they continue to show any eye changes.

Prognosis

Prognosis has always to be most guarded. Infection in some kennels will decimate the inhabitants whilst in others the majority will recover under similar treatment. There is evidence to suggest that other factors besides infection *per se* are involved to bring about the full clinical picture of H.P. disease. The protean nature of some outbreaks also, should make one hesitate over prognosis. I can illustrate this point by three cases which occurred in my kennels last year. A Dachshund bitch, aged four years, was admitted on "heat." On entry she appeared normal, but developed hysteria four days later. Reference to the owner elicitated the fact that three months previously she had had a bout of coughing for a week, followed by several attacks of hysteria, but had apparently recovered. She was removed forthwith to an isolation kennel, where she rapidly developed hard pads and acute encephalomyelitis, and was destroyed. In the boarding kennel next to her was a six months' old poodle. This cheerful pup was noticed to be quiet one morning—temperature 106°, which fell back to normal within 24 hours. Penicillin was continued for four days, but as temperature remained level, no more was done. Six days from the primary rise, this pup suddenly developed hysteria and was dead within an hour. *Post-mortem* examination revealed extensive haemorrhage over the cerebral hemispheres. Laboratory report positive to H.P.

The third case, an Alsatian bitch aged three years, after the usual primary phase symptoms, developed acute pneumonia and cardiac distress; she looked certain to die, yet made a comparatively quick recovery on penicillin. Laboratory reported blood positive to H.P. The infection in this outbreak started from the Dachshund, which was virtually a "carrier," spread to the poodle next door, and then to the Alsatian, and yet there is little similarity between them clinically and the prognosis almost impossible to make accurately.

When puppies are infected during the teething period, if they recover the dentine is found to be severely injured—so-called "distemper teeth."

* Presented to the Sussex Division, N.V.M.A., at Brighton, February 18th, 1949.

Treatment

Owing to the very varied course that this disease can take, it follows that it is difficult to assess with any degree of accuracy how efficacious any line of treatment actually is.

I am convinced that there is one essential and that is absolute and complete rest for a minimum of three weeks from the primary rise and that this period must be extended if the sclerotics continue to be injected.

At the present time, on first seeing a case, I inject 20 c.c. H.P. serum and put the animal on to penicillin-in-oil injections. I repeat 10 c.c. H.P. serum weekly as long as eye discharge is present. In-contacts receive 10 c.c. H.P. serum. Aspirin dissolved in potassium citrate solution is used as a routine mixture.

No sulphonamides in any form are prescribed. If diarrhoea is not severe I give Greenosan (two tablets) crushed up in milk. For some months now I have changed the diet back to raw egg and milk in small quantities ; also sweetened tea. The enteric symptoms are often most difficult to control. From a brief trial of Aerosporin, I feel hopeful that we have a medicament that will deal effectively with the Gram-negative organisms that seem to flourish in these cases.

About 18 months ago I tried a series of cases on injections of Anahaemin with little success, but in view of A. L. Duckett's reports in *The Veterinary Record* (December 4th, 1948), I feel that I may possibly have been using this liver preparation too late. At the moment I am trying Duckett's suggestion, using "Plexan" in the early stages to see if I can obtain results comparable to this.

Discussion

Of the various schools of thought on the aetiology of H.P. disease, which I mentioned earlier, I belong, naturally, to that group which regards the condition as being largely due to a virus that has been with us for many years and has frequently been mixed up with, or mistaken for, L. and D. infection. I say "naturally" simply because I have maintained openly[3], and for a good many years, that on clinical and pathological grounds such a virus existed and was, in fact, responsible for the brain lesions found in so-called "breakdowns" to L. and D. inoculations—and yet, although I had seen hard pads in days before, I never realised their pathological significance until my attention was drawn to this point.

The fact that the simple injection of H.P. virus into a normal non-immune dog produces only a thermal reaction, and that in naturally occurring outbreaks there are many non-clinical cases comparable to the above, suggests that there are some other factors necessary within the host for the full development of the main features of H.P. disease. There is, for example, a good deal of clinical evidence that the early use of sulphonomides can bring about changes within the body which will predispose the host to develop the disease in all its evil features.

In this connection, Duckett's recorded observation, when discussing the treatment of H.P. with "Plexan" liver extract, is interesting. He stated, "If administered early the liver treatment will not prevent the pads developing a fairly marked degree of hardness, *but it does give from 40 to 50 per cent. recoveries in all but the very young.*" If this observation is substantiated in a good series of cases, it would not only make a definite advance in treatment but would suggest at the same time another approach to the study of the pathology of this condition, since "Plexan" might fairly be described as a mitrailleuse of the B.2 complex, the component factors of which are known.

At the commencement of this paper I said I should be brief in order to obtain the fullest possible time for discussion. I will conclude, therefore, on this note—and I told you I was going to be provocative—that it is my firm belief that if a safe technique for immunising dogs against H.P. virus, as well as L.D. virus, can be devised, then field or street distemper will have been stripped of most of its terrors for me. By the same token, fulfilment of this work on the H.P. virus must inevitably lead to an advancement in knowledge of the pathology of those distressing diseases of man associated with demyelination. To this end, therefore, we, as clinicians, must see to it that we exert our best endeavours to help research workers to attain this goal, and complete the work so well begun by MacIntyre, Trevan, and Montgomerie at the Wellcome Research Laboratories.

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Opening Contribution to Discussion

By

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I hasten to offer my most sincere congratulations to Major Pugh on his very excellent and concise contribution on this all-important subject to those concerned in dog practice. It has been a privilege to listen to him. He has provided us with plenty of material for discussion, but in his characteristically modest way, has made no more than passing reference to his former work upon encephalitis. I feel that his paper to-day should be read in conjunction with those which he published in 1926 and 1935 under the titles "A Preliminary Note on the so-called Dry Distemper or Infectious Brown Mouth of the Dog[1]" and "Endemic Encephalitis in the Dog [1A]." In these he describes a series of cases which, apart from some minor differences, correspond very closely with the disease which we to-day know as "hard pad disease." I understand that experts have examined sections made, and have found them indistinguishable from sections taken from recent cases of hard pad disease. This adds considerable weight to his thesis that the virus of hard pad has been present in this country for a long period and that from time to time it has become manifest, as during the present pandemic. Another pointer is that in the past some dogs suffering from what was thought to have been canine distemper were noticed to lick the feet. This was explained as being a type of neuritis associated with the disease. Is it not possible that it was actually due to the pain associated with hardening of the pads? Licking of the feet is, of course, a common symptom in hard pad.

When I first read Major Pugh's assertion made at the Southport Conference that, since hard pad has been so widespread, ordinary distemper has been comparatively uncommon, I frankly disagreed. Since that time, however, this statement has become borne out by our experience in the Guildford area and I have been forced to amend my views. During the summer, except for outbreaks of hard pad at certain kennels, neither disease was particularly prevalent, but latterly we have encountered many cases of hard pad and, in fact, a major epidemic is in progress, while canine distemper, on the other hand, has become comparatively uncommon.

In my opinion, the difficulty of differentiating between canine distemper and hard pad in the early stages of these diseases is the crux of the whole problem at the moment. If the sick dog has been inoculated against distemper it is a fair assumption that this disease is not involved, but where no immunisation has been carried out, a diagnosis in time for the use of either serum to be of any avail, is extremely difficult. How easy it would be if some simple test-tube examination could be evolved giving a positive diagnosis in one or other disease. It has been stated that the proliferative changes in the keratogenous layers of the pads commence early. I should be grateful if one of the research workers could state how early in the disease these changes actually commence. I appreciate the difficulty of the technique that would be required, but do they consider that examination of a very small section taken from one of the pads would indicate the presence of hard pad virus at an earlier stage than do clinical symptoms? Even in the case of a dog that has been immunised against distemper, diagnosis is not always easy, for cases are frequently seen showing what appear to be typical early symptoms of hard pad, but which clear up in a few days with or without treatment. The question arises, were these dogs actually affected with hard pad without secondary infection, or was some other agent responsible? I raise this point because it is such cases which make the assessment of the value of hard pad serum difficult, for if recovery occurs immediately after the use of serum one is naturally inclined to assume that it was the serum that brought about the cure. This may be so in many cases, but I am afraid that one often claims credit for the serum when it is not strictly entitled to it! Response to the use of serum (and we have used a considerable quantity in the Guildford practice) has been somewhat variable, and at times a little disappointing. As Major Pugh has pointed out, the age at which a dog contracts the disease has a definite bearing upon the prognosis. It may be this age factor and the inherent resistance of the patient which influence to a large extent the success or otherwise of the use of serum. The length of time that the virus has actually been present in the body is, of course, a factor of the utmost importance, but this aspect will be touched upon later.

The first case upon which I used serum with success was in a ten-year-old wire-haired terrier bitch, weighing about 22 lb. The animal was seen on Saturday, September 11th, two days after it first went off its food, and its temperature was 104°. The breathing was rapid and there was a slight husky cough. Sulphapyridine was prescribed, and the animal was seen again on the following Monday. There was no improvement in the condition, and at this point 125,000 units of penicillin were injected, and repeated

twice daily, but still there was no apparent improvement. By Thursday an intense conjunctivitis had developed with engorgement of the vessels of the optic sclera. This we now recognise as an almost characteristic sign of hard pad. An opacity of the cornea was developing. During Friday and Saturday the temperature remained slightly elevated, and on Sunday, September 19th, ten days after the first symptoms were noted, there was definite evidence of hardening of the centre of the pads, together with an increased roughness round the edges. Thirty c.c. of hard pad serum were injected intravenously that evening. The following day the animal seemed brighter, but the temperature remained elevated until Tuesday evening. On Wednesday morning it was normal and the appetite returned. From that point progress was rapid, and the pads began to peel on the following Monday, and at the time of writing, nearly five months after the illness, the bitch is in good health, with no evidence of nervous symptoms. It will be noted that a second dose of serum was not given, but since this case we have learnt to our cost that it is unwise to rely on a single injection.

I know that I am going contrary to the accepted theory, and contrary to the experimental evidence presented by MacIntyre, Trevan and Montgomerie[2] when I say that we have not noted and marked undesirable sequelae following the use of a sulphonamide in cases of hard pad. The explanation of this may be in the fact that whereas these workers gave full therapeutic doses for eight days, we continue administration for three days only. In several cases the use of sulphonamides has been avoided, but no significant difference in the incidence of nervous complications has been noticed. I should like to ask Major Pugh what he considers to be the explanation of the phenomena which both he and other workers have noted, following the use of sulphonamides.

When I was preparing these notes I intended quoting in detail the case of a wire-haired terrier which appeared to make a classical response to the use of hard pad serum in December, 1948. Last week, however, the animal showed weakness in the hind quarters, and on Sunday became quite blind, and whined continuously. It was destroyed the same evening. Careful enquiry elicited the fact that a week or two before the injection of the serum, the dog had shown symptoms of a mild malaise, accompanied by a slight temperature. It is evident that this was the real beginning of the illness.

Major Pugh stressed the great variation that may occur between different cases on the same premises, and I endorse his observations fully. In very general terms, there appear to be, in our area, two distinct syndromes. The first goes through the stages such as one sees in a typical distemper. The other is the case in which the premonitory symptoms are so slight that they pass unnoticed, even if, in fact, they have been present at all. The dog will have a fit, or a series of fits which may become almost continuous, and it dies, or has to be destroyed shortly afterwards.

We know that the incubation period of hard pad in ferrets can be from six to 32 days, but can Dr. Montgomerie or his co-workers tell us what is the range of incubation in the dog, and how long the virus may be active in the body before obvious clinical signs are manifest? An interesting case came to our notice a few months ago which may have some bearing upon this question. A six-months'-old Samoyed dog came into the surgery on August 7th, 1948, to be immunised against distemper. It had a temperature of 104° and, as this did not subside after a period of rest, it was decided that it would be unwise to carry out the inoculation, in spite of the fact that the owner was insistent that the dog was quite normal in every other respect. It was brought in again on September 1st, but again its temperature was 104°, and although the dog was kept quietly at the surgery all day, its temperature did not fall. A three-day course of M. & B. 693 was prescribed. The dog remained apparently quite normal until September 16th, one month after being first seen, when it had a fit. On the 20th it had a second fit, and four days later diarrhoea commenced. On September 26th there was evidence of hardening of the pads. Early in October he started to whine almost incessantly, and by the 9th of the month appeared to have become mental. Keratosis of the nose was marked by this time. The dog was destroyed on the following day. It would appear from this case that the virus may be active in the animal's body, as indicated by the persistent temperature reaction, for a very considerable time before there is any outward sign that it is ill. Had the dog been inoculated, there is little doubt that the owner would have suspected that the immunisation had been responsible for the condition.

I have already mentioned the difficulty one often encounters in deciding whether or not one is dealing with hard pad, and although I will not go into great detail, this difficulty was well illustrated in the case of a kennels in which an outbreak of illness occurred. The dogs immunised against distemper appeared to resist the infection but distemper serum did not prevent the development of symptoms in the other young dogs, nor did large doses effect a cure. In retrospect, it is quite evident that the whole outbreak was caused by the hard pad virus but at the start a definite diagnosis presented the utmost difficulty. The first two dogs were negative but ferrets inoculated with material from the third dog to die developed symptoms of hard pad after 24 days. There was thus a considerable time lag before a definite diagnosis was possible, especially as the characteristic hardening of the pads was absent from the early cases. To illustrate this point, I am sure that Mr. MacIntyre will forgive me if I quote him as having said upon receiving two sick puppies, that he considered them to be suffering from ordinary canine distemper, as they had typical symptoms, with no hardening of the pads. The ferrets inoculated with material from the puppies, however, developed hard pads in from seven to 20 days. Cultures from all the organs yielded *Salmonella enteritidis* in pure culture. Hard pad serum first became available at the time when these puppies were ill, and they were each given an injection of 20 c.c. intravenously, but it was too late to have any appreciable effect upon the course of the disease. In spite of this serum, however, hard pad virus was still present in the tissues as shown by ferret inoculation. This adds weight to Major Pugh's assertion that it is necessary to repeat the serum at intervals. How long would Mr. MacIntyre consider that the virus would remain active in the body in an untreated case?

I should like at this point to place on record my very sincere appreciation of the very ready help and co-operation we have received from Dr. Montgomerie, Mr. MacIntyre and their staff in carrying out *post-mortem* and cultural examination on the not inconsiderable number of dogs that have been sent to him. He must have been inundated with specimens from all over the country, and yet he has been able to furnish a detailed report upon each case sent from our practice.

Nearly all the cases submitted had pneumonia, and the following organisms were cultured from the lungs and/or other organs: *B. coli, B. proteus, Salmonella aertryke* and *S. enteriditis*. Most of the cases had been treated with penicillin and/or M. & B. 693 (there appeared to be no significant difference between the incidence of nervous symptoms between the two groups). It is obvious that it is no longer the Gram-positive organisms that are our chief concern as secondary invaders, but the Gram-negative group.

Now Major Pugh has mentioned the use of an anti-biotic called "Aerosporin," and I am glad to hear that from his brief trial he considers it to be of value. This substance, according to Brownlee and Bushby[3] is more discriminating in its activity than streptomycin, and is from ten to many hundred times as active as the latter in inhibiting certain coli-typhoid-dysentery organisms and including pasteurella and salmonella infections. It does not readily produce resistant strains. I should like to draw attention to a few other anti-biotics which, although not necessarily available in this country at the moment, will probably become so in the future, and may have a useful action against the secondary organisms associated with hard pad. A substance known as "Aureomycin," obtained from *Streptomyces aureofaciens*, and which has recently received considerable publicity in the press, should be closely followed. It is claimed[4, 5] that it has a very wide therapeutic spectrum, being active against both Gram-negative and Gram-positive organisms *in vitro*. Amongst other organisms, it is said to be active against the following: Haemolytic streptococci, staphylocci, *B. coli, Haemophilus influenzae, Brucella abortus* and *Brucella suis*. What is even more important, Aureomycin is active against certain viruses. It has been used in man against the virus causing lymphogranulomata, and may be given both orally and parentally. An anti-biotic having rather similar properties to Aureomycin is "Chloromycetin"[6]. Other substances are "Streptothrycin," "Grisein," and, of course, "Streptomycin." The last two appear to have some synergistic action, and when combined tend to reduce the tendency to set up resistant strains of some bacteria.[4]

The prevention of infection with hard pad would, of course, as Major Pugh has stated, be a tremendous step forward, and I am anxious to know from Dr. Montgomerie whether he is hopeful that a method of immunisation may be evolved at a not-too-distant date. It would, of course, be ideal if it could be successfully combined with the distemper inoculation. Major Pugh ended his paper with a statement that, if a technique for the immunisation against hard pad is evolved, distemper would be stripped of most of its terrors. Would he care to elaborate this theory?

In conclusion, I should like to say how much I appreciate having been asked to open the discussion on Major Pugh's paper this afternoon. I only hope that I have not taken up too much of the short time available for discussion of so interesting and important a subject.

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General Discussion

Dr. David Trevan: It would be possible to examine specimens of pad if these could be obtained without doing too much damage to the dog. They should be about 1 mm. thick, right through the epithelium and including a piece of connective tissue. The section should be cut during the first four to six days. This material could be injected into susceptible dogs.

Hard pad was difficult to reproduce in the laboratory and contacts to artificial hard pad did not show the symptoms observed in the field. They had a fever about six days afterwards which subsided within a week, and five weeks later hard pad virus was obtained from these animals. Their blood gave hard pad disease to the ferret, but they never had hard pads and they never had fits. Sulphonamides in very high doses were given, by mouth, for a week about the same time the virus was injected. A week later some had fits and others developed hard pad.

He agreed with Major Pugh in thinking that there must be some other factor involved, and hoped something would arise during the meeting to give some clue to that other factor. He said it was certain that dogs could carry the virus while appearing perfectly normal, and mentioned an instance of typical hard pad disease which recovered while in the kennels. Two months after recovery its blood infected ferrets with hard pad disease. He thought it probable that sulphonamides did not have any effect on the course of the disease. The incubation period was from one week to two months.

Mr. Weipers, of Glasgow, had suggested that dogs were only infectious in the very early stages of the disease. It was a fact that there were diseases, such as encephalitis of dogs, which appeared very similar to hard pad disease. There were, however, certain minor pathological differences so far absent in the other cases. There were serological differences between the virus of distemper and that of hard pad, and there was now undoubtedly a lack of dogs with the classical distemper.

Mr. Balls: He could only speak of the disease as he understood it in his own experience. He had found marked symptoms of pain, both peripheral and abdominal. In some instances it was so acute that they almost looked like foreign body cases. It had sometimes led to a good deal of hard searching to find whether the cause was a foreign body or hard pad disease. Some had actually had foreign bodies, were operated upon and developed hard pad disease not long afterwards. He wanted to know more about hard pad serum, how early it could be used, and how late. He asked if it was of any use if nervous symptoms had already started: he had tried it in one case in which nervous symptoms had begun and it had not had any effect. He had also given serum to dogs in contact with a known case of hard pad disease and they had developed fits 10 to 14 days later.

Mr. Scott Dunn: Hard pad disease did not exist in his practice as it did in others. In his regular work at the greyhound stadium he had not seen hard pad disease in a greyhound. He pointed out that Sealyhams and Bedlingtons were often found to have a thickening of the pads and wondered if some of the cases diagnosed as hard pad disease had had hard pads for a long time.

Mr. Peace: He had never seen hard pad disease and was not quite clear what it was. In hysteria and fits in young dogs the possibility of hard pad disease was not examined. Not so long ago many cases which appeared to be nervous distemper would not have been examined for hard pad disease—they were destroyed.

Col. Turner: He had a very wide experience of greyhounds and had never seen hard pad disease in that breed. He supported Mr. Balls concerning abdominal pains in dogs which afterwards developed distemper or hard pad disease.

Major Lawrence: He agreed with Major Pugh's description of the symptoms, but queried Mr. Crawshaw's statement that anorexia was usual. His experience had been that in the majority of cases there was no lack of appetite. On the contrary, the majority of cases had a good appetite, even when they went crazy. He felt that the disease had existed for a long time as he remembered treating similar cases as long ago as 1940, but with nothing like the same frequency as at present. The disease had now reached serious proportions and was a great menace to the dog population and the small-animal practitioner. He wondered if it was possible that the virus had mutated from the true virus of Laidlaw and Dunkin as he had not seen a case of old-fashioned distemper for some time. He had had only a few cases of the disease among dogs which had been inoculated against the Laidlaw and Dunkin virus by the Field method, and such cases appeared to have a better chance of survival. He asked what chance there was of incorporating protection against so-called hard pad virus with the ordinary anti-distemper inoculation? He also enquired if anyone had cured an animal which had developed nervous complications, such as fits, chorea or paralysis? In his opinion the name of "hard pad disease" was bad because it was misleading (as the pads did not become noticeably hard until the dog had been ill for some time) and it was unscientific. He suggested that a more accurate and more scientific name should be found for the disease and thought that the old name of "Continental distemper" was a much better name than "hard pad." He felt that a great deal of harm had been done by inaccurate newspaper articles and comments, chiefly by over-stressing the importance of the lesion of hard pads. He suggested that the N.V.M.A. should do something to check the wrong impression given by the press and also to educate the public to the seriousness of the disease and the necessity for early treatment. The difficulty in practice was to get the cases sufficiently early to effect a cure. Usually they were presented when the dog had been ill for two or three weeks or even longer and by that time the virus was "fixed" and the brain cells damaged, and he could not see how the specific serum could be expected to do any good. To be effective it must be given within a week of the onset of the disease.

Dr. Plunkett: In relation to human disease, he was particularly reminded of instances where sulphonamides were given for dysentery, etc., in such large doses that they produced a toxic effect; the cure was large doses of nicotinic acid. The condition seemed to be caused by destruction of bacterial flora in the intestines to such an extent that the gut became sterilised and synthesis of vitamin B was inhibited. Taking the disease as a whole, it reminded him of the South American pellagra, now regarded as an avitaminosis. In that disease there was diarrhoea, sometimes bloodstained, dermatitis and dementia, with hyperkeratosis, followed by desquamation and peripheral neuritis. Negro women were particularly affected. According to the locality, a variety of vitamins were lacking. Crude liver extract had been effective but folic acid had been shown to be far more effective. Riboflavin had also been used. The disease could last for extremely long periods. He thought hard pad might be acute encephalitis plus vitamin deficiency. As greyhounds were fed on large quantities of eggs and appeared to be immune, he wondered if eggs afforded any protection. Also, was it not possible that the virus of hard pad was normally present in dogs and only produced the disease when the animal was in a low state or suffering from avitaminosis?

Mrs. Kon did not think the disease had anything to do with sterilisation of the gut because sulphonamides were not given in large enough quantities. She wondered if it was possible that the harmful effects of sulphonamides were due to the knocking out of para-animo-benzoic acid.

Mr. Willett had noticed that chorea could occur from the pure Laidlaw and Dunkin virus, and wondered if all these cases of chorea were hard pad disease. Regarding diarrhoea, sometimes bloodstained, sometimes not, he found it cleared very satisfactorily with sulphamethazine. Symptoms were sometimes not associated with hard pad disease at all. Concerning the late use of serum, he had seen a four-year-old Scottie on the last day of January which appeared to have typical hard pad disease. The owner reported that it had been boarded out over Christmas. The animal's eyes were extremely red at that time but otherwise it appeared normal. He had given 20 cc. of hard pad serum and, for the diarrhoea (not bloodstained), sulphamethazine for four days, at the end of which time it had ceased. A very small area at the end of the tongue was necrotic and sloughed off. The dog had improved rapidly, the eyes cleared, and it now appeared normal.

Miss Phillips: The first instance of actual hard pad disease which she remembered was in 1943 and she had not seen another until 1945. She felt that dogs' feet were seldom examined, and several cases might have been overlooked. She wanted to know what percentage of clinical cases actually had hard pads. She found that diagnosis was very difficult. She asked Dr. Trevan at what stage blood samples should be taken or if the virus could be found throughout. In dealing with the disease from 1943 to date, she found that in the earlier outbreaks tonsillitis was the first symptom, but in present-day outbreaks diarrhoea was the more common. Before the war, dogs were found with streptococcal sore throat, which never showed nervous symptoms and which responded to sulphonamides. She found little true distemper in her district. She asked how long animals could continue being carriers and how infection was transmitted. Did Major Pugh recommend the administration of 20 c.c. of serum to all breeds, irrespective of size, or should it be given on the basis of 1 c.c. per lb. body weight? She had noticed that there was very marked oedema of the throat after

the use of serum. On one occasion she had given 150 c.c. to a St. Bernard and oedema had been enormous. She asked Dr. Trevan how long passive immunity lasted with hard pad serum and whether he had any idea what factor caused the difference between hard pad disease in the laboratory and in the field.

Miss Uvarov: She had been depressed rather than helped by the discussion so far. She realised that, serious though it was that the dog population was suffering, it was more serious that the veterinary surgeon was suffering at the hands of the lay press and of clients. She thought it would be better if the name "hard pad" could be suppressed as the number of cases in which there was actually thickening of the pad was slight. The name "Continental distemper" also was too vague. She, personally, told her clients that the dogs had catarrhal fever and told them what it might be, simply to safeguard herself. She saw cases of hardening of the pads; they were sometimes shiny and slightly painful and, later, hard. It was not a condition often seen in greyhounds, but she had seen it in two Irish greyhounds. She had only seen one case of hard pad in the R.S.P.C.A. clinics or in animals belonging to poor clients. Out of about a hundred to two hundred dogs seen in a week there were quite a number of instances of catarrh, but only one of hard pad. There was one thing which she did find a slight help. After the fourth or fifth day, if the eye was examined with an ophthalmoscope, minute haemorrhages were found in the fundus of the eye which did not appear in other diseases. She thought it best to take clients into her confidence and to explain the cost of serum treatment. She had found that many cases of the dogs with hard pad were those which had been inoculated against distemper, and wondered if it was coincidence or if, by inoculating the animal against distemper, something else was disturbed. When hard pad serum was scarce she had not used it in desperate cases, but had given a dose of anti-distemper serum, and used hard pad serum when she thought there had been a better chance of recovery—the proportion of recoveries had been about the same. For the secondary conditions she never used sulphonamides, but vitamin B in the form of either brewer's or baker's yeast. She had observed no beneficial results from the use of liver extract.

Mr. Grant: In May and June, cases suffering from fever and pharyngitis, which recovered after non-specific therapy, all returned in October suffering from hard pad. He found serum 50 per cent. effective therapeutically. He had found, in several cases where serum had been used after fits had started, that nembutal, intravenously, had been reasonably successful. Following vaccination against distemper, a number of litters of puppies had shown intermittent diarrhoea three weeks after inoculation. He had given hard pad serum and, with no further treatment, the diarrhoea ceased and the pups recovered. In future, he intended to give hard pad serum at the time of distemper vaccination to check this. He felt members should approach the liaison officer to obtain some publicity in the press. Dog owners should be made aware of the use of serum and of its dangers.

Mr. Ingram thought the secondary consideration which brought hard pad into existence was probably nutritional. Dogs were now fed on salvaged knacker meat and he wondered if this had any bearing, since there was now more Salmonella infection in dogs. He agreed that after inoculation for distemper a certain number of dogs did develop hard pad disease. He gave one example of a dog which had been inoculated against distemper. Within a week it had developed continuous fits, and was destroyed. Another animal had been given anti-distemper serum as soon as diarrhoea appeared; the diarrhoea had not improved, and this dog was also destroyed.

Mr. Chipperfield wanted a new name for the disease. He felt "canine encephalitis" was preferable to "hard pad."

Dr. Montgomerie: There were people who said that the Wellcome Laboratory had invented the disease as an excuse for the failure of their distemper serum! The origin of the name arose from laboratory language. In true distemper breakdowns there were symptoms which were a true distemper picture and distinguishable from hard pad disease. Some work had been done during the past year. He was now confused rather than befogged. There was an entity which the profession had been calling hard pad disease; it was due to a virus which was not the same as Laidlaw and Dunkin distemper virus. So far as diagnosis was concerned, some progress had again been made. Dogs inoculated against Laidlaw and Dunkin virus had some immunity to hard pad disease. If he and his colleagues were not so overcome with specimens at the laboratory they would have more time to apply to this point. Another advance had been the production of the serum which had some value in treatment and some prophylactic value, though they had not yet made very much advance with active prophylaxis, but this was being pursued. Dr. Trevan had pointed out one thing which had unexpectedly been of value: this was the natural infection of hard pad disease in ferrets. Within a very short time of arrival at the laboratory some ferrets had developed hard pad disease. He could not understand people who declared that hard pad disease did not exist. He stated that the use of sulphonamides was definitely contra-indicated, as they interfered with the bacterial flora and produced an antibaccinosis. He recommended the use of vitamin B in large doses; as much as 20 mg. had been given intravenously.

Dr. Trevan asked for the carcases of Bedlingtons and Sealyhams which died from any cause. He wanted to know where distemper had gone. Up to 1945 there had been encephalitis due to distemper and different from hard pad disease. Dogs were known to recover after showing nervous symptoms, though not very often. There was some hard pad disease immunity after the administration of prophylactic doses of serum. He agreed that hard pad was a bad name for the disease but did not know what else it could be called. Practitioners had been asked to try vitamin B for treatment of the condition, but great care was essential in its administration as it could be dangerous in excessive doses.

In reply to Miss Phillips, he said that six weeks was the length of passive immunity in contact dogs and that blood samples should be taken at the height of febrile reaction.

The Reply

Major Pugh: The name "hard pad" seemed to be worrying most people. About 20 years ago he had suggested the name "epidemic encephalitis in dogs" for this condition and could see no objection to its use, as it described the main features of the condition. Although there had been well marked outbreaks in Kent in 1924, 1925 and 1928 right up to the outbreak of war, the present outbreak appeared to have cleaner cut features than before and the use of penicillin and sulphonamides might have contributed to this end. He was most interested in Miss Uvarov's point about eye haemorrhages and intended to look for this point in future. Dr. Plunkett had suggested that the sulphonamides might interfere with the synthesis of the B_2 group and there seemed to be some supporting evidence for this suggestion.

Regarding the acute abdominal pain mentioned by several people, he suggested that this was really spinal in origin. The cord lesions in some of these cases were most profound. In answer to the question "How early should serum be given?" he said that it should be given as soon as a case was seen and repeated weekly. Replying to Mr. Scott Dunn's doubt concerning the actual existence of hard pads, he said that there was not the slightest doubt on this point. He had seen cases in which the hardened areas had gradually loosened off the pads and made a clinking noise on hard surfaces.

He quoted an interesting case of chorea following distemper which had remained static for 2½ years. The dog had then contracted hard pad and the choreiform movements rapidly spread till the whole body became involved and the dog was destroyed. Such cases as these seemed to support those who contended that there were two different viruses at work. If a safe technique for immunising against both distemper and hard pad disease could be evolved, he could feel much happier about distemper inoculations.

THE FILM AS AN AID TO THE PRACTICAL APPLICATION OF SCIENCE

"The important part which the film has to play in making scientific discoveries 'live' in practical terms to the actual worker using the new processes was the theme of the first conference of its type ever held in Scotland," reports the *Cinema* in its issue of June 8th, and continues: "It was held at the Royal (Dick) Veterinary College and organised by the Scientific Film Association. The conference showed twelve films on agricultural subjects. These sought to demonstrate in practical condensed form the basic work of scientists engaged on work of vital importance to agriculture. Attendance consisted of farmers and young agriculturalists from all over Scotland.

"The theme of the gathering was set by Professor Henry Dryerre, who said that one of the greatest problems of the present time was to pass on to the practical farmer the increasing store of scientific knowledge. The enormous discoveries in laboratories and in field practice were piling up in places to which the ordinary farmer had no access. He thought that films could assist materially in bridging that gap."

The Scottish Film Council co-operated with a display of film equipment.

CLINICAL COMMUNICATION

Otodectic Mange in the Dog

E. KAUFMANN AND CHURCHILL FROST

HAMPSTEAD

We have believed for some time that otitis externa in the dog is very often caused by *Otodectes cynotis*. In consequence we have recorded our findings in the last 50 consecutive cases of otitis. In each case the auricular discharges and debris were examined microscopically, and in many cases the external auditory canal was examined with an auroscope. Five of the dogs had trouble with one ear only and all were found to be unaffected with ear mange. Of the 45 dogs with both ears affected, a total of 28 were demonstrated microscopically to be caused by parasitic otitis (*Otodectes cynotis*). With the aid of the auroscope one could see the movement of the parasites in the ears of several of the dogs.

Symptoms described by the owners of the patients varied from frequent shaking of the head, rubbing the ears on the floor and excessive scratching, to owners not noticing any ear trouble.

Facts and deductions concerning the 28 positive cases we record numerically below:—

1. The youngest puppy was aged three months.
2. The oldest dog was 5½ years of age.
3. Seventeen (60 per cent. approx.) dogs were under nine months old.
4. The majority of the 17 were fairly certain to have been affected at the time of purchase. Also the puppies were probably affected at the time of weaning. This suggests a maternal source of contagion, but we have no proof of this in any of the recorded cases.
5. In two instances two dogs belonging to the same owner were affected. Infection from one dog to another is probable in one pair aged five and 5½ years respectively. The dogs came from different sources and had not lived with any cats.
6. Three dogs living in close contact with cats proved to be affected. Several more dogs were associated with cats. It is probable that cats are a common source of infection to the dog.
7. The breeds affected were: Eight Poodles, six Dachshunds, three Pekingese, three Spaniels, two Scotties, two Yorkshire terriers, one Labrador Retriever, one Corgi, one Alsatian, and one Chow.

Dachshunds and Poodles appear to be most commonly affected, although there were three poodles amongst the 22 dogs not proven to be suffering from ear mange.

8. Other external parasitic troubles present in some of the dogs were fleas, lice and demodectic mange. (No case of sarcoptic mange.)
9. All cases have responded to treatment with Ascabiol (May & Baker, Ltd.).

SUMMARY: 56 per cent. of 50 cases of so-called canker in the dog proved to be caused by otodectic mange and responded to specific treatment.

Acknowledgments.—We wish to thank Dr. J. N. Oldham for confirming the species of our type specimen.

ABSTRACTS

[Studies on Vehicles for Sustaining Penicillin Levels in the Bovine Mammary Gland. FOLEY, E. J., STULTS, D. W., LEE, S. W., & BYRNE, J. V. (1949.) *Amer. J. vet. Res.* **10.** 66.]

The authors describe a series of experiments to study the comparative ability of two specially formulated water-in-oil emulsion vehicles, A and B, to maintain a satisfactory level of penicillin in the bovine mammary gland and to compare them with that of an aqueous vehicle. Quarters of non-infected cows in full lactation, infected cows in full lactation and dry cows were used in the experiments. The vehicles were first of all proved to be non-irritant to both dry and lactating cows. A single injection of 100,000 units penicillin in 20 cc. water and in the two vehicles under trial, showed that levels of the drug were maintained for significantly longer periods with vehicles A and B than with water, indicating that the properties of the vehicles used are of decisive importance in maintaining levels of penicillin in quarters in which it is instilled. With vehicle B, effective penicillin levels were maintained for 72 hours following a single intramammary injection of 100,000 units. An experiment is also described in which a tracer dye, trypan blue, was used to demonstrate visually the remarkable degree of dispersion obtained with vehicle B compared with an ordinary aqueous vehicle. This result offers an explanation for the impressive prolongation of the penicillin level, described in the first part of the paper.

The significance of these results in relation to the treatment of *Streptococcus agalactiae* infection is discussed and a valuable list of references is included.

J. A. N.

* * * * *

[Differentiation of Acid-fast Bacteria by means of Serological Tests. WESSELS, C. G. (1948.) *J. S. Afr. vet. med. Ass.* **19.** 45-57.]

Protein extracts were made from nine strains of acid-fast bacilli obtained from skin lesions, seven other strains of acid-fast bacteria, *viz., M. tuberculosis bovis, hominis, avian, phlei, butyricum* and two strains of so-called leprosy bacilli.

Rabbits were immunised with purified specific proteins prepared from these strains and the properties of their sera were studied. The author showed that there was no close relationship between the saprophytic acid-fast bacilli and the mammalian tubercle bacilli. He therefore concludes that the former will not sensitise to mammalian tuberculin. The skin lesion bacilli appeared to be serologically homogenous. They differed fairly widely from mammalian tubercle bacilli but had some antigenic relationship to avian tubercle bacilli and to the leprosy bacilli.

The significance of these findings is somewhat reduced because the purified protein extracts were made after growing the bacilli on Long's medium, and it is the general consensus of opinion that neither true leprosy bacilli nor skin-lesion bacilli have been cultivated on any laboratory medium.

J. F.

National Veterinary Benevolent and Mutual Defence Society

ANNUAL GENERAL MEETING AT MANCHESTER

The annual general meeting was held at the Midland Hotel, Manchester, on May 10th, 1949, with the President, Mr. John Holroyd, in the chair.

Apologies for absence were received from Messrs. J. Adamson, C. W. Cartwright, H. W. Dawes, H. J. Hodgman, W. R. McKinna, R. C. Locke and A. J. Wright.

The minutes of the last annual general meeting were read and confirmed.

Chairman's Remarks.—Mr. HOLROYD referred to the work covered during the year and took pride in the substantial reserve funds. In these days, however, it was impossible to estimate the extent of any one claim on the Defence Fund and he would not be satisfied until a reserve of at least £10,000 had been created. The Benevolent Fund was sound and he urged members to report any cases of distress or difficulty which might come to their notice.

ELECTION OF OFFICERS

President.—In proposing the re-election of Mr. John Holroyd as President, Mr. SUMNER referred to the magnificent work he had done and was still doing for the Society. The proposition was seconded by Mr. A. H. WATSON and carried unanimously.

Vice-Presidents.—Mr. H. Sumner and Mr. A. H. Watson were re-elected as Vice-Presidents on the proposition of Mr. J. HOLROYD, seconded by Mr. H. W. STEELE-BODGER.

Council.—The following members were elected as Council Members: Messrs. J. Adamson, Junr., C. W. Cartwright, H. W. Dawes, O. V. Gunning, F. Beckett, D. E. Leask, R. C. Locke, W. R. McKinna, P. T. Lindsay, T. A. Mitchell, Mrs. E. H. Shaw, Messrs. R. F. Ross, C. F. Shawcross, H. M. Wilson, H. W. Steele-Bodger, W. K. Townson, A. J. Wright and T. Wilson.

Hon. Secretary-Hon. Treasurer.—Major F. Hopkin was re-elected as Hon. Secretary and Hon. Treasurer on the proposition of Mr. HOLROYD, seconded by Mr. SUMNER, and it was resolved that there should be recorded in the minutes of the meeting the deep sense of appreciation felt for Mr. Hopkin's work over a long number of years.

The Hon. Treasurer's Report and Accounts were adopted as circulated.

The Hon. Secretary's Report was adopted as circulated.

It was proposed by Mr. STEELE-BODGER, seconded by Mr. LEASK and agreed that the annual report and accounts and the minutes of the annual general meeting be sent to the *Record* for publication.

It was agreed that future lists of names of members should be prepared so as to differentiate between members of the Defence Section and members of the Benevolent Section.

The Hon. Secretary's Report

This Report was as follows:—

Defence Fund.—The building up of an adequate Reserve continues, but the Executive have a high minimum in mind, having regard to the high values of animals ruling at the present time, and the possibility of even one claim running into several thousand pounds. For this reason, the addition of many practising veterinary surgeons as new members is welcomed, but the Council take this opportunity of restating that the Defence Fund is available to members for the purpose of defending them, or indemnify them where necessary, against claims arising from the commission or omission of some act in the course of the treatment of or attendance on any animal. The cover does not include losses by fire nor does it include any liability ordinarily covered by a Third Party policy of insurance. This rule is restated, as cases referred to the Society during the year, which were obviously outside the scope of the Defence Fund, have included (1) objection by neighbours to barking and howling dogs in nearby kennels; (2) claim by farm worker for personal injury from a hypodermic needle; (3) possible slander cases. Needless to say, the Society has given what help and advice it could in these cases.

Fifteen cases were dealt with during the year. Of these, four claims were admitted and paid by the Society in the total sum of £395 0s. 2d., plus costs. Five claims totalling £1,573 were successfully defended or resisted. In two cases, no claims resulted and the remaining four cases were either outside the scope of the Defence Funds or were not pressed.

Members are reminded that it is necessary to renew membership every year, and renewal forms are sent out in December. It will greatly assist the work of the Hon. Secretary and Treasurer if members would give these forms their prompt attention to avoid the issue of reminders.

Benevolent Fund.—Donations to the Fund have helped to improve still further the financial strength of the Fund and thanks are due to all contributors. Grants made total £519 15s. and all members, and particularly new members, are asked to co-operate by reporting to the Society any case in which they feel the Benevolent Fund can be used to relieve distress among members of the veterinary profession or their dependants.

. . . . . .

The National Veterinary Defence Society—Defence Fund

Statement of Affairs as at January 1st, 1948

Liabilities	£ s. d.	*Assets*	£ s. d.
Sundry Creditors, per list	127 10 8	Cash at Bank ...	2,275 13 0
Capital Account ...	2,148 2 4		
	£2,275 13 0		£2,275 13 0

Income and Expenditure Account for the Year Ended December 31st, 1948

Expenditure	£ s. d.	*Income*	£ s. d.
To		*By*	
Claims Paid	395 0 2	Subscriptions and Entrance Fees received ...	2,557 2 6
Legal Charges re Claims	27 6 0	Subscriptions for Students	187 3 0
Insurances of Students ...	111 6 6	Donations received—	
Printing, Stationery, Postages, etc.	53 6 9	Society of Veterinary Practitioners ...	250 0 0
Hire of Rooms	6 4 0	T. A. R. Chipperfield, Esq.	10 6
Secretary's Salary ...	100 0 0	Bank Interest	5 10 0
Audit Charges	18 18 0		
Bank Charges	2 0 0		
Income Tax	18 0		
	714 19 5		
Balance to Capital A/c...	2,285 6 7		
	£3,000 6 0		£3,000 6 0

Balance Sheet as at December 31st, 1948

Liabilities	£ s. d.	*Assets*	£ s. d.
Sundry Creditors ...	68 1 3	£2,000—3% Defence Bonds at Cost ...	2,002 18 0
Capital as per last Balance Sheet ... £2,148 2 4		Cash at District Bank ...	1,498 12 2
Add Balance from Income and Expenditure A/c 2,285 6 7		Cash at Knutsford Savings Bank	1,000 0 0
	4,433 8 11		
	£4,501 10 2		£4,501 10 2

The National Veterinary Benevolent Society—Benevolent Fund

Receipts and Payments Account for the Year Ended December 31st, 1948

Receipts	£ s. d.	*Payments*	£ s. d.
To		*By*	
Balance at District Bank, Ltd.	219 4 2	Purchase of £100 2½% Consols	81 1 0
Redemption of Manchester Corporation £300 Bond, No.19828	300 0 0	Purchase of £300 3% Savings Bonds ...	299 4 6
Premium for Conversion of 3% Defence Bonds to 2½%	3 0 0	Purchase of £400 3½% War Loan	414 6 6
Income from Investments, per Schedule	392 12 11	Insurance of Victory Bonds against Redemption	6 0 0
Donations as per Schedule	244 8 3	Grants	519 15 0
Amount recovered from Income Tax Reclaim, 1947/48	238 10 2	Bank Charges	3 6 8
Members' Subscriptions...	72 9 0	Balance at District Bank, Ltd.	146 10 10
	£1,470 4 6		£1,470 4 6

Donations Received, year ended December 31st, 1948

	£ s. d.
Executors of Mrs. Clancy (Legacy)	101 4 3
Anonymous	100 0 0
Lancashire Veterinary Association	7 19 0
Association of State Veterinary Officers	5 5 0
Midland Counties Veterinary Medical Association ..	5 5 0
Yorkshire ,, ,, ,, ..	5 5 0
Lincolnshire ,, ,, ,, ..	5 0 0
Mrs. Elmes	4 4 0
Spalding Services Welcome Home Fund	4 0 0
A. Shenton, Esq., ...	2 2 0
G. Dickinson, Esq.	2 2 0
J. J. Davies, Esq.	10 6
C. G. Hopson, Esq.	10 6
T. D. M. Martin, Esq.	10 6
W. T. McGregor, Esq.	10 6
	£244 8 3

ROYAL ZOOLOGICAL SOCIETY OF SCOTLAND

Commenting at the annual meeting of the Royal Zoological Society of Scotland on the immense improvement that has taken place during the past year in the collection on view at the Zoological Park at Corstorphine, Professor James Ritchie, Vice-President, said that the pre-war standard had now nearly been reached. In the last two years the Society had spent nearly £3,000 on the animals. They could spend more on animals and they had the offer of animals, but the Council had been restrained because they could not build the accommodation for them owing to the difficulty of getting labour and materials.

Referring to the schemes the Council at present had in hand, Professor Ritchie said that one of them was due to a grant of £1,000 from the Pilgrim Trust to provide for the proper labelling of the cages so that the animals might easily be identified. A second scheme was for a water circulation in the ponds in the lower part of the park and was making progress. This, when completed, would enable those ponds to have one or two complete changes of water every 24 hours.

Professor Ritchie also referred to a proposal to inaugurate a health service at the park with a veterinary officer and a proper hospital in which sick animals could be attended. It was hoped to interest the Veterinary Research Association in the matter.

Councillor R. E. Douglas made an appeal for a wider circulation for the new magazine, *Scottish Zoo and Wild Life*, which was at present being run at a loss.

* * * * *

SOIL EROSION DANGERS

The dangers of poverty and malnutrition through soil erosion and methods of prevention are explained in "Alphabet of the Soil," the second pamphlet in the Unesco series, "Food and People," published by the Bureau of Current Affairs. The pamphlet is written by Dr. Guillermo Nannetti, a Colombian professor of sociology and a member of the executive board of Unesco.

* * * * *

Weekly Wisdom

". . . Mankind was now in the trough of waves, and in the horrors of that experience should be more watchful than ever before, for that was the time when things went wrong. . . ."—General Smuts, speaking in London on June 7th, 1949.

The Supplementary Veterinary Register

First List of Applicants for Registration

We publish below a notice which is being issued by the Royal College of Veterinary Surgeons in the case of each individual applicant for registration on the Supplementary Veterinary Register.

In our issue of May 21st, 1949, we explained at length the procedure by which registration should be applied for by unqualified persons, and again we particularly draw attention to the letter which was written to each applicant by the Royal College of Veterinary Surgeons in sending the form of application for registration. This sets out clearly some of the rather abstract points in connection with registration.

We would again call attention to our leading article on the subject of the Supplementary Veterinary Register in our issue of May 21st, in which we pointed out that the Council of the Royal College will be in a judicial position in considering these applications. Justice must be helped and we would again emphasise that where any veterinary surgeon has any information regarding any of the applicants in the list given below he should, if it is material, communicate with the Royal College.

Again, we would emphasise that it is a duty to do so, not only in the case where registration is not justified but also in the case where it is within the personal knowledge of a veterinary surgeon that an applicant is entitled under the Act to registration.

Whatever the feelings of the profession may have been in the past about unqualified practice it is necessary and it is honourable that the abolition of unqualified practice should be carried out with scrupulous fairness.

VETERINARY SURGEONS ACT, 1948

SUPPLEMENTARY VETERINARY REGISTER

RESTRICTION OF PRACTICE OF VETERINARY SURGERY BY UNQUALIFIED PERSONS

NOTICE IS HEREBY GIVEN that the persons set out below, resident at and working at the addresses given below their names, and being persons without a veterinary qualification registrable on the Register of Veterinary Surgeons, have applied for registration on the Supplementary Veterinary Register.

The conditions for registration on the Supplementary Veterinary Register are that an applicant has attained the age of 28 and satisfies the Council of the Royal College of Veterinary Surgeons:—

(a) that he is of good personal character;

(b) that during the ten years immediately preceding the application he has for a period of not less than seven years in the aggregate been engaged in the United Kingdom as his principal means of livelihood in diagnosing diseases of animals and giving medical or surgical treatment to animals:

Provided that the Council may authorise the registration of a person under this section who has been engaged as aforesaid during the said ten years notwithstanding that he has been so engaged for less than seven years in the aggregate if the Council are satisfied that the time during which he was so engaged was reduced by reason of his serving in His Majesty's forces or being engaged in work of national importance and that his registration would not be prejudicial to the public interest.

Any person having any OBJECTION to the registration of any applicant and desiring to lay any information before the Council thereon should communicate with the undersigned at the Royal College of Veterinary Surgeons, 9/10, Red Lion Square, London, W.C.1, not later than 30 days after the publication of this notice, provided that an extension of the period for objection may be allowed if good cause be shown for such extension.

W. G. R. OATES,
Registrar.

VETERINARY SURGEONS ACTS, 1881-1948.

THE FIRST LIST OF NAMES OF PERSONS WHO HAVE APPLIED FOR REGISTRATION

Number	*Name and Address*	*Place of Practice*
S.V.R. 43	ADAM, N. J. (Miss), The Loft, Hale Place, Farnham, Surrey.	None.
104	AUSTIN-SMITH, W., 182, Blackfen Road, Sidcup, Kent.	The same address.
131	BARNETT, W. H. E., 5, Thorpe Street, Old Trafford, Manchester.	26, Oxford Road, Manchester, 16, and 7, Broad Street, Salford, 6.
79	BATES, B. C. (Miss), The Cottage, 85, Main Road, Burton Joyce, Notts.	P.D.S.A., 25, Carlton Road, Nottingham.
68	BEAL, H. J., 82, The Bayle, Folkestone, Kent.	The same address.
71	BILLINGTON, T. H., 9, Northcote Road, Strood, Kent.	7, Guil Lane, Strood, Kent.
39	BLAKE, P. D., Chiltern Canine Infirmary, Longwick, Aylesbury, Bucks.	The same address.
63	BROWNE-COLE, R., "Brookthorpe," Nailsea, Somerset.	The same address and 63, Wells Road, Bristol, 4.
34	BROWNE-COLE, W., 127, Cheltenham Road, Stokes Croft, Bristol, 6.	The same address and 583, Gloucester Road, Horfield, Bristol; also 45, Whiteladies' Road, Clifton, Bristol.
98	BROWN, L. T., 83, Windermere Avenue, Elm Park, Essex.	P.D.S.A., Woodford Bridge Road, Ilford, Essex.
62	BURNS, T. J., 29, Waverley Road, St. Albans.	65a, St. Peter's Street, St. Albans.
10	BYERLEY, M. R., 58, Sydenham Park, London, S.E.26.	56, Greenwich South Street, London, S.E.10
77	CAREY, E. G., 1, Elm Grove, Gloucester Road, Bath.	The same address.
115	CLARK, M. (Mrs.), Flat 3, 15, Marine Square, Brighton, 7.	P.D.S.A., 3, Montague Place, Brighton, 7.
27	CLEVELAND, W. H., 53, London Road, Halesworth, Suffolk.	The same address.
36	CONNORTON, C. G., 510, Chessington Road, West Ewell.	316, Balham High Road, S.W.17; and the same address.
70	COOMBS, E. R., 8, Calvert Terrace, Swansea.	The same address.
6	COOPER, C. F., 3, Ledbury Road, Croydon, Surrey.	The same address.
60	COOPER, R. J., Redberry, Abberton, Colchester.	The same address.
90	CRONIN, J. T., 58, Herent Drive, Ilford, Essex.	P.D.S.A. Sanatorium, Woodford Bridge Road, Ilford, Essex.
164	DRINKWATER, A., Brunswick House, Belle Orchard, Ledbury.	The same address.
64	EDWARDS, E. P., Bellamy's House, Brinkworth, Nr. Chippenham, Wilts.	The same address.
106	FOULSTON, W. F. N., Aintree House, Keelby, Habrough, Lincs.	The same address.
20	FLETCHER, H. J., 354, West Barnes Lane, New Malden, Surrey.	The same address.
46	GANNON, A. B. J., Westerley, Tyndale Park, Herne Bay, Kent.	215, High Street, Herne Bay, Kent.
102	GRAINGER, W. A., 5, Cairnside, East Herrington, Sunderland.	The same address.
9	GRAY, E. V. G., 98, Manor Road, Wallington, Surrey.	The same address.
37	HALL, I. G., Lyndhurst, St. John's Street, Huntingdon.	The same address.
41	HARRISON, J. M., 63, North-Eastern Road, Thorne, Nr. Doncaster.	The same address and Epworth, Nr. Doncaster.
54	HATCH, F. R., 69, Battersea Rise, S.W.11.	The same address.
58	HAYES, C. H., 40, Coombe Road, New Malden, Surrey.	The same address and 100, London Road, Kingston-on-Thames.
51	HENSON, G. H., 33, Crouch Hill, London, N.4.	The same address.
35	HILLARY, B. L. P., 9, Roy Road, Northwood, Middlesex.	The same address and 1, Station Road, Northwood; also 33a, College Road, Harrow, Middlesex.
52	HINDLE, R., 2, Railway Approach, Chertsey.	The same address.
13	HOPKINS, J., 271, Osmaston Road, Derby.	The same address.

Number	Name and Address	Place of Practice
S.V.R. 5	HUGHES, I., 55, High Street, Saltley, Birmingham.	The same address.
35	INGRAM, G. L. (Miss), Victoria Cottage, Clyde Street., Kirn, Argyll.	The same address.
42	JAMES, J. C., Whitehall Wick, Dursley, Glos.	The same address.
40	JONES, F. H., Evedale, 1, Palace Avenue, Rhyl.	The same address.
89	KING, F. G., Park Villa, Crudwell, Wilts.	The same address.
112	LEE, G., 736, Barking Road, Plaistow, E.13.	The same address.
75	LLOYD-JONES, W. L., The Denes, Station Road, Preston Park, Brighton, 6.	The same address and Palorit House, 11, Harrow Road, W.2.
134	LOWRIES, J. E. (Mrs.), The Lodge, Woodhaw, Egham, Surrey.	The same address.
25	MARGARSON, E. T., The Bungalow, Mundford, Thetford, Norfolk.	The same address.
65	MARKING, R. A., 2, Morocco Street, London, S.E.1.	The same address and Sandown Paddocks, Epsom, Surrey.
38	MUMFORD, R. F. J., Hurst Lodge, 326, London Road, Waterlooville, Hants.	The same address.
28	MURCH, C. T., Hazelcroft, Church Road, Plymstock, Devon.	The same address.
91	MURES, A. A., 37, Linden Avenue, Whitstable, Kent.	27a, Middle Wall, Whitstable.
140	MURRAY, J., Preyste House, Petworth, Sussex.	The same address.
44	NEWSTEAD, A. E., 33, Osborne Road, Broadstairs, Kent.	The same address and 22, Albert Street, Ramsgate.
8	PLANTON, G. F., 5, The Quadrant, Redland, Bristol.	P.D.S.A., Bristol.
121	POOLE, L. J. H., Henley Bank Kennels, Brockworth, Glos.	The same address.
94	POWELL, B. M. Grenville Lodge, Newport Pagnell, Bletchley, Bucks.	The same address.
12	READ, H. S. M., 377, Wickham Road, Shirley, Croydon.	The same address.
15	REYNOLDS, E. H. B., Main Road, Rocklands, Attleborough, Norfolk.	The same address.
33	RHEASON, W. P., 102, Ashbourne Road, Mitcham, Surrey	25, Grenfell Road, Mitcham, Surrey.
105	RICHARDSON, H. R., Market Hill, Hedon, Nr. Hull.	The same address and 107, Coltman Street, Hull.
47	ROBINS, M. W., Hassocks Orchards, Hassocks, Sussex.	The same address.
108	ROGERS, S. J., 15, Douglas Road, Hounslow, Middlesex.	The same address.
53	RUSSELL, H., 12, Ironmill Place, Crayford, Kent.	The same address and 156, Windmill Street, Gravesend, Kent.
66	SEGGAR, L. C., 89, Argyle Road, Sheffield, 8.	123, Chesterfield Road, Sheffield.
73	SCOTT, H. S., 158–160, Chingford Mount Road, South Chingford, E.4.	The same address.
7	SHERLOCK, F. J., Pennisfield Farm, Silver Street, Irlam, Lancs.	The same address.
56	SHIPSTONE, C. H., 147, High Street, West Wickham, Kent.	147 and 149, High Street, West Wickham, Kent.
92	SMITH, F. J., 27, Lower Street, Hillmorton, Rugby.	The same address.
17	STEAR, W. C., Vine Cot, Aveton Gifford, Kingsbridge, Devon.	The same address.
29	STOREY, J. C., 70, Gladstone Road, Wimbledon, S.W.19.	17, Granville Road, Southfields, S.W.8.
32	THOMAS, J. J., Penybont, Llansawel, Llandilo, Carms.	The same address.
S.V.R. 156	WADDELL, B. C. F., The Retreat, Buckingham Park, Shoreham, Sussex.	199, Albion Street, Southwick, Sussex; also 16, John Street, Shoreham, Sussex.
100	WAGNER, H., Liberty Hall, Brookshill, Harrow Weald, Middlesex.	382, Station Road, Harrow, and the same address.
101	WAGNER, V. M. (Mrs.), Liberty Hall, Brookshill, Harrow Weald, Middlesex.	The same address and 382, Station Road, Harrow.
83	WALL, G., 114, Wavertee Road, Liverpool, 7.	The same address.
118	WALTON, J. S. R., 262, Wylds Lane, Worcester.	The same address.
113	WHITE, L. W., 10, Mellows Road, Woodford Avenue, Ilford, Essex.	P.D.S.A. Sanatorium, Woodford Bridge Road, Ilford, Essex.
22	WILKINSON, E. (Mrs.), 163, Chorlton Road, Manchester, 16.	The same address.
11	WILSON, C. G., The Kennels, Medstead, Alton, Hants.	The same address.
96	WINGFIELD, W., 22, Whitehall Road, Halesowen, Worcs.	The same address and 418, Bearwood Road, Smethwick, Staffs.
3	WORROW, H., 4 Redlands View, North Holmwood, Dorking, Surrey.	The same address.

NOTES AND NEWS

Diary of Events

June 30th.—Meeting of the North Wales Division, N.V.M.A., at Siamberwen, Llangollen, 2 p.m.

July 15th.—Meeting of the Mid-West Division, N.V.M.A., at Bristol (Berkeley Café, Clifton), 2.15 p.m.

July 15th.—Second Annual Meeting of the Association of Veterinary Teachers and Research Workers (Southern Region), at the Royal Veterinary College, Camden Town, N.W.1, 2 p.m.

Aug. 8th to 13th.—Fourteenth International Veterinary Congress, Central Hall, Westminster, and Church House, Westminster, London, S.W.1.

Aug. 12th to 15th.—Sixty-seventh Annual General Meeting and Congress, N.V.M.A., in London.

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XIVth International Veterinary Congress

Professor A. Verstraete, Chairman of the Ghent Veterinary School, supplements his recent note on the Belgian delegation with the pleasing information that a subscription has been entered by the Agricultural High School of Ghent (affiliated to the Universities) and that, in addition to Professor L. Geurden, the Minister of Agriculture has also appointed as delegate Professor Ch. van Goidsenhoven, Rector of the Veterinary School of Cureghem (Brussels). Professor Goidsenhoven will also represent his School, while Professor G. Peeters will be the official delegate of the Veterinary School of Ghent.

Dr. D. M. Campbell, Editor of *Veterinary Medicine* (Chicago), is conducting a tour of delegates and other visitors from the United States, and Dr. J. V. Lacroix, Editor of the *North American Veterinarian*, writes: "It should be of interest to European veterinarians to learn that Dr. Carl F. Schlotthauer and Mrs. Schlotthauer plan to make this trip. Dr. Schlotthauer has been on the staff of the Mayo Clinic for just over 25 years. Veterinary practitioners who keep themselves informed are acquainted with some of Dr. Schlotthauer's work. He has been rather a prolific writer, he is an indefatigable worker, and his activities embrace the field of general practice as this pertains to diseases of horses, cattle, swine and dogs. In addition, Dr. Schlotthauer has given special attention to canine surgery. His work in the Mayo Foundation keeps him in touch with all sorts of research wherein dogs are employed for subjects, by Fellows who work at the Mayo Foundation under the direction of the staff men at the Mayo Clinic, and others."

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PERSONAL

Colonial Veterinary Service Appointments.—The Colonial Office announces the following promotions and transfers in the Veterinary service: Mr. R. W. Crowther (Veterinary Officer, Cyprus), Veterinary Research Officer, Cyprus; Mr. H. T. B. Hall (Government Veterinary Officer, Fiji), Deputy Director of Agriculture

(Veterinary Services), Jamaica; Mr. L. R. Hutson (Veterinary Officer, Barbados), Chief Veterinary Officer, Leeward Islands; Mr. C. W. Wells (Veterinary Officer, Federation of Malaya), Chief Veterinary Officer, Singapore, and Mr. D. G. White (Senior Veterinary Officer, Tanganyika), Assistant Director of Veterinary Services, Tanganyika.

The appointment also is announced of Miss M. N. Massie, M.R.C.V.S., as Veterinary Officer, Tanganyika.

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Birth.—JENNINGS.—On June 9th, 1949, at Liverpool, to Isobel Winifred Jennings, M.R.C.V.S., wife of A. R. Jennings, B.V.SC., M.R.C.V.S., Liverpool, a daughter.

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Marriage.—NOBLE—HARPER.—The marriage arranged between Claude Noble, M.R.C.V.S., of Woodbank, Crossgates, Fife, and Christine Harper, of 8, Avondale Road, Wolverhampton, took place quietly on June 2nd, 1949, at St. Jude's Church, Wolverhampton. The best man was Vernon Clark, M.R.C.V.S., of Walsall.

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Central Society's forthcoming visit to Newmarket.—For their Summer Outing this year, on Thursday, July 21st, the Central Veterinary Society will be visiting Newmarket to see the work being carried out at the Stations of the Animal Health Trust. Full particulars will be announced in our Notices column, shortly; meantime, Fellows are asked to note the date, as it is hoped that there will be a large attendance for this specially interesting event.

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R.C.V.S. OBITUARY

WHITE, Munro Erskine, "Cherry Croft," Francis Way, Salisbury, Wilts. Graduated Glasgow, April 1st, 1896. Died June 16th, 1949, following a long illness; aged 74 years.

PAGE, Charles Wood, F.R.C.V.S., Deddington, Oxford. Graduated London, March 29th, 1883; Fellowship April 28th, 1892. Died at Banbury, June 10th, 1949; aged 88 years.

MR. C. WOOD PAGE, F.R.C.V.S.

Mr. Wood Page, whose death we record above with much regret, spent a lifetime in the practice at Banbury now run by Mr. H. C. Shingler. He was, in fact, born on the premises at 47, The Green, and took over from his father. Needless to say, he was one of the old horse lovers, taking very reluctantly to the motor-car. Mr. Page had been retired since 1930 and, sad to say, bedridden for nearly two years.

THE LATE COLONEL F. C. STRATTON

Colonel F. C. Stratton, whose death we had the sad duty of recording in our last issue, was a great sportsman. A racehorse owner, he had the satisfaction of winning many events of standing, while he was also a very keen fisherman and golfer. A retired officer of the R.A.V.C., he served many years in India. Colonel Stratton had resided with his sister at Babbacombe since 1939.

The funeral service was held at Little St. Mary, conducted by the Vicar (Rev. J. C. L. Johnstone) and Rev. W. F. G. Wittey. Cremation at Plymouth followed. Among those present at the service were Lt.-Col. Bartlett (representing Brigadier Plunkett and officers of the R.A.V.C.), Mr. F. J. Bonnor, Mr. H. G. Rowbotham (representing the captain, directors and members of Torquay Golf Club), Mr. F. M. Buckland (representing R.T.Y. Club) and Mr. C. R. Buswell (representing lady captain Mrs. C. R. Buswell, Torquay Golf Club).

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DEATH OF DR. HERMANN MIESSNER

We regret to learn of the death, which took place on May 4th, of Dr. Hermann Miessner, Emeritus Professor of Microbiology and Veterinary Hygiene at the Hannover Veterinary College.

Professor Miessner taught in Hannover from 1912 to 1939 and became well known abroad through his participation in the preparation of several textbooks of international repute.

Professor Miessner was repeatedly elected Chancellor of the Hannover Veterinary College and held this post during the College's celebration of its 150th Jubilee in 1928.

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Interdepartmental Committee on Meat Inspection

This committee, the Minister of Food's intention to appoint which was announced in our issue of June 11th, is composed of officials from the Ministry of Food, the Health Departments in England and Wales and in Scotland, and the Ministry of Agriculture and Fisheries. Mr. G. R. Oake (Ministry of Food) has been appointed Chairman and the other members are:—

Ministry of Food: Messrs. H. R. Barnell, M.A., B.SC., L. B. A. Grace, M.R.C.V.S., W. A. Lethem, M.D., W. J. M. Menzies, H. D. Vigor, O.B.E. *Ministry of Health*: Messrs. P. N. R. Butcher, E. L. Sturdee, M.R.C.S. *Department of Health for Scotland*: Mr. Howat. *Ministry of Agriculture and Fisheries*: Mr. J. Cameron, M.R.C.V.S.

The Committee's terms of reference are:—

(i) To review the memorandum on a system of meat inspection known as Memo.62/Foods;

(ii) to consider whether Memo.62/Foods, revised as necessary, should be given statutory authority in new Food Regulations superseding the Public Health (Meat) Regulations, 1924-48, for England and Wales and corresponding legislation for Scotland;

(iii) to consider whether the inspection of home-killed meat at the place of slaughter can be made compulsory in England and Wales as it is already in Scotland, and whether this requires any further statutory authority;

(iv) to consider whether the Public Health (Meat) Regulations, 1924-48, require revision in any other respects;

(v) to consider the qualification of those at present engaged in meat inspection and the qualifications which should be required of those so engaged and the facilities for their training;

(vi) to consider the existing requirements and methods of operation, as regards imported meat, under the Public Health (Imported Food) Regulations, 1937-48,

and to make recommendations to the Minister of Food, the Minister of Health, and the Secretary of State for Scotland.

Mr. W. J. B. Hopkinson (Ministry of Food) has been appointed Secretary to the Working Party and to the Inter-departmental Committee. All communications should be addressed to him at 47, Portman Square, London, W.1.

MEAT PRODUCTS WORKING PARTY

In the same issue we announced the appointment by the Minister of a Meat Products Working Party, which has as its chairman, Mr. S. W. Hood, C.B., and, among the assessors (representing the Ministry of Food), Mr. L. B. A. Grace, M.R.C.V.S., D.V.S.M., the Ministry's Chief Technical Adviser on Meat Inspection.

The Working Party's terms of reference are: To review present trade practice and legal requirements for securing that conditions in the meat manufacturing trades are clean and sanitary and that the products, and the materials from which they are prepared, are wholesome in all respects; to draw up, if considered desirable, agreed codes of practice for voluntary adoption by the trades concerned; to consider whether new or amended requirements by way of statute or regulations are desirable, and to make recommendations to the Minister of Food, the Minister of Health, and the Secretary of State for Scotland.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

SHEEP SCAB:

Yorks (West Riding).—W. H. Wearmouth, Heugh Farm, Eastby, Skipton, Yorks. (June 14th).

* * * * *

Care of Laboratory Animals.—A discussion on this subject will be held at noon on Tuesday, June 28th, at the Grand Hotel, Harrogate. Professor R. J. V. Pulvertaft, the president of the section of pathology and bacteriology of the British Medical Association, will take the chair. Professor A. N. Worden, M.R.C.V.S., and Dr. Jean Vinter will speak on behalf of the Universities Federation for Animal Welfare.

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A Conference to discuss the best means of promoting an expansion in pig production is being arranged by the N.F.U. for July 13th. Representatives of all organisations interested in every aspect of pig production, marketing and distribution are being invited.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible.

CONTROL OF TRYPANOSOMIASIS IN AFRICA

REFUTATION OF CONCEPTION OF CONTINENT AS "A SOURCE OF MEAT SUPPLY"

Sir,—Captain W. W. Henderson's remarks in his letter in your May 7th number are accurate and factual as regards animal disease and its control in Africa. Even Dr. D. G. Davey seems to have

toned down the original very optimistic prophecies that antrycide would mean the end of animal trypanosomiasis—see the report of his address to the joint meeting of the Royal African Societies in London, where he warns against the production of resistant strains and that prophylactic experiments had shown that there were certain obvious difficulties. Captain Henderson's appeal for urgent research to combat contagious bovine pleuropneumonia is apt and important.

With his views on the export of meat from Africa I disagree profoundly and suggest that all available facts show that Africa suffers from acute meat shortage. The appalling meat deficiency in West Africa was stressed by Professor W. C. Miller in his report of 1946 on the Veterinary Dept. of the Gold Coast, where the amount of meat available for the local native was incredibly small. The position in Nigeria, where my friend Captain Henderson spent so many useful years, is not so bad but even there the vast mass of the population is grossly meat deficient. The Union of South Africa and Southern Rhodesia are disease-free compared to tropical Africa and yet these countries do not produce adequate meat for their small white population, while the native masses have mealie meal as their staple diet and meat as an occasional luxury, but compared to tropical Africa meat is abundant in Southern Africa. If ever the indigenous population of Africa is to approach to physical efficiency, then every scrap of meat and meat products grown in Africa will be required in that continent and then more will have to be imported. Prior to the recent war the Gold Coast was the largest importer of meat and meat products in the British Empire except for the Mother Country alone. Fortunately the Gold Coast is a comparatively "well-off" colony and the favoured "upper ten" of the Africans in the cocoa belt could afford to buy canned and salt meats. In many parts of Africa everything animal which dies, except hyenas and vultures, is eaten because of the terrible shortage of animal protein. African grazing carries fewer animals to the area than other continents and there are huge areas of well populated tropical forest and wet lands which literally carry no livestock at all.

In the past year I have read similar optimistic prophecies that Africa is to feed Britain, by public personalities headed by such an eminent oracle as Sir Stafford Cripps, and it is time that such wishful thinking is contradicted. I am surprised to find such an authority as Captain Henderson kicking against the pricks of hard facts. His peroration, however, suggests that his judgment has been clouded by the meagre meat ration of Kilbride compared to his past abundance at Vom. I rejoice that I have abundant meat in the wilds of the Northern Transvaal, but it is indubitable that were supplies equally divided among the entire population of this province and including the depressed natives, Captain Henderson would be a meat glutton at Kilbride when compared to myself at Magoeba's Kloof.

Yours faithfully,

Magoebaskloof, J. L. Stewart.
Northern Transvaal.

June 9th, 1949.

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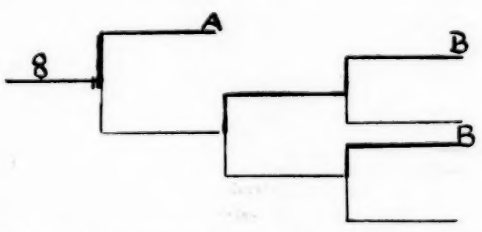
EVIDENCE OF AN INHERITED SEMINAL CHARACTER ASSOCIATED WITH INFERTILITY OF FRIESIAN BULLS

Sir,—With reference to the article in *The Veterinary Record* of May 28th, 1949, by J. L. Hancock, concerning an hereditary seminal characteristic associated with the head of the sperm of Friesian bulls, we should like to add further evidence in support of his findings.

Recently, we examined a Friesian bull, aged 21 months, which was sterile, and enquiry revealed that its breeding was similar to those recorded by Hancock. Using the method shown in Hancock's article, the breeding can be presented as follows:—



The bull was first used at the age of nine months, and over a period of a year had given 34 services to five cows and 15 heifers; none of the services has proved fertile. Two examinations of the semen were made at an interval of two weeks and a total of five ejaculates was obtained. On both occasions the abnormality of the sperm head was visible during the examination of the fresh preparation on the farm. In contradistinction to Hancock's findings, the motility and viability of the semen were subnormal, but it was also noted that, on palpation, the testes were approximately three-quarters normal size.

For morphological examination, smears were made of undiluted semen and stained with haemalum and eosin. Duplicate smears were prepared with 10 per cent. nigrosin. Owing to the distance from the laboratory, the smears were not prepared and stained until some hours after collection, when it was apparent that the number of affected sperms was lower (16 to 42 per cent.) than in the bulls examined by Hancock. As a further test,[1] smears were made at 24 and 96 hours after collection, and it was found that the number of affected sperms had decreased from 30 per cent. at 24 hours to 10 per cent. at 96 hours. In view of the work of Blake[2] in New Zealand concerning hereditary sperm defects, the tracing of further cases of the type recorded is of considerable interest.

Yours faithfully,

Veterinary Investigation Department, D. H. L. Rollinson.
Upper Redlands Road, J. B. Makinson.
Reading, Berks.

June 2nd, 1949.

References

1 Hancock, J. L. Private communications.
2 Blake, T. A. (1945.) *Nature*, **155**, 631.

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FINANCIAL CONTROL IN THE NEW COUNCIL, R.C.V.S.

Sir,—During the three years when I was President of the Royal College of Veterinary Surgeons I was much concerned with the number of letters which appeared in *The Veterinary Record* on various points concerning the Veterinary Surgeons Acts, the Council of the Royal College, and matters incidental thereto, and which have contained either mis-statements of fact or have shown misreading of acts, regulations, or other matters vital to the veterinary profession.

In your issue of June 4th there appeared a letter from Mr. Arnold Spicer headed "Financial Control in the New Council." In view of the fact that certain misconceptions may arise out of this letter I think it necessary to make the following points quite clear.

The Veterinary Surgeons Act, 1948 (not the new charter, as Mr. Spicer states, because the new charter has not yet been granted and in any event has nothing to do with annual fees), permits the Council to prescribe an annual fee not exceeding five guineas. It should be carefully noted, a point which Mr. Spicer's letter ignores, that before any annual fee may be prescribed it must be confirmed by order of the Privy Council; in other words, no increase in the annual fee could be made unless there was good reason for such increase to be made and the Privy Council were satisfied as to the reason.

Mr. Spicer's suggestion that the Treasurer and all members of the Finance Committee should be drawn only from the elected members of Council would appear to be a most undesirable aspersion on all appointed members of Council, whether the appointment be made by His Majesty's Privy Council, universities, or veterinary colleges, and whether the appointed members be veterinary surgeons or laymen. It is most unfortunate, and indeed derogatory to the dignity of the profession, that there should be any attempt at the present time, or at any time, to create a rift between elected and appointed members of the Council.

The study of the working of legislation concerning professions regulated by Parliament shows that there is appointed representation on the Councils governing other professions (for example the General Medical Council and the Dental Board) and in other professions the bodies concerned have had the good taste not to discriminate amongst their members whether elected or appointed.

The principle underlying the appointment of one or more lay persons to the statutory council of a profession is to ensure that there should be in each such council the voice of one or more persons of experience and standing outside the professional circle who can, where necessary, present an outside point of view.

Before Mr. Spicer's letter had ever appeared the Council (which has a majority of elected members) had chosen the Finance Committee, which consists of one M.P. and 19 veterinary surgeons, and of these 19, 14 are elected members of the Council.

There is thus one lay person on the Finance Committee and five appointed members who are veterinary surgeons; all these five were previously elected members of Council. If Mr. Spicer's sug-

gestion be correct then all these five, by the sheer reason of their appointment, will no longer have any interest in the welfare of the veterinary profession and in the spending of its money. Speaking for myself, who has been an elected member of Council for 15 years prior to my appointment by the Privy Council, and who has been a practitioner for a lifetime and will continue to be a practitioner although an appointed member, I can only regard Mr. Spicer's suggestions with amazement.

Mr. Spicer's letter would, appear to suggest further, that the finances of the R.C.V.S. are in the sole control of the Treasurer and the Finance Committee, whereas the truth is that both the Treasurer and the Committee are responsible to the full Council, which I need scarcely repeat again has a majority of elected members.

Mr. Spicer's suggestion that the office of Treasurer be confined to the elected members of Council is also unfortunate when coming from a former member of the Council. The present Treasurer—Professor J. B. Buxton—was elected to that office whilst an elected member of the former Council. The new Council *unanimously* re-elected him at the recent Council meeting although his status on the Council had changed to that of an appointed member.

As in all matters requiring common sense and ensuring the benefit of the profession, the answer is that the persons best fitted for an office or for membership of a committee must be selected whatever their exact status may be; to suggest otherwise would be an aspersion on the elected members themselves.

I trust that the above remarks may make the position quite clear to all members of the profession and destroy the very unfortunate and erroneous impression which may have been given by Mr. Spicer's letter.

Yours faithfully,

Camden House, H. W. Dawes.
West Bromwich,
Staffs.
June 20th, 1949.

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MEAT INSPECTION: INTERDEPARTMENTAL COMMITTEE

Sir,—Your issue of June 11th, 1949, brings the welcome news that the Minister of Food has decided to appoint an "Interdepartmental Committee" to look into the requirements of meat and food hygiene.

Although the composition of this committee is unknown,* one does not hesitate to believe that veterinary representation will be included as four major functions in public health, *i.e.* (i) the control of animal diseases transmissible to man; (ii) meat inspection; (iii) inspection of food of animal origin, and (iv) milk sanitation, are based on veterinary science and closely connected with the veterinarian's professional training.

Where the veterinary surgeon has not been deprived of his natural prerogatives but on the contrary has been given full active participation in the control of such fundamentals as meat, milk and food of animal origin, the results achieved speak for themselves (America, Germany) and are worthy of praise both from an economic and public health point of view.

The failure to take full advantage of the veterinarian's knowledge and co-operation in the field of meat and food hygiene in this country is a deplorable fact. A splendid opportunity has now arisen to rectify this grave mistake of the past. A veterinary branch added to the existing public health service would improve the hygiene of food processing, wholesomeness and quality of meat and food of animal origin and would help to protect not only the consumer's health but his pocket as well.

Yours faithfully,

Department of Veterinary Hygiene, A. Ginsberg.
Royal (Dick) Veterinary College,
Edinburgh, 9.
June 13th, 1949.

* As intimated elsewhere in this issue, the composition of the committee has since been announced; it includes two members of the veterinary profession in Messrs. J. Cameron, M.R.C.V.S., and L. B. A. Grace, M.R.C.V.S.

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CATTLE ON HILL FARMS IN THE HIGHLANDS AND ISLANDS

Sir,—So far as I can ascertain, milk fever, difficult calving, infertility, mastitis and abortion are not met with in cows on hill farms where the calves are reared according to the laws of nature.

Cattle from these farms are in demand at auction sales, as buyers know they are hardy and improve rapidly when transferred to better land.

If tuberculosis were eliminated from these herds and more attention paid to breeding the herds would be a first-class source of supply for dairy and fattening animals for farmers who do not rear their own stock.

If part of the millions spent overseas were transferred to hill farms and marginal land there would soon be a great increase of home-produced beef and less reliance placed on foreign sources.

Yours faithfully,

The Anchorage, A. Hart.
Ardnadam, Dunoon,
Argyll.
June 16th, 1949.

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PERSONNEL OF THE PROFESSION

Sir,—All those sincerely concerned about the future of the profession will have followed with interest the expressions of opinion which have appeared as the result of Professor McCunn's letter of March 15th.

Mr. G. H. Arthur contends, I think correctly, that our main task lies with the health of farm animals, and the general trend of veterinary education suggests the future graduate will be better fitted for this work.

Will these people find themselves redundant? Professor McCunn asks. I would say "yes"—unless, of course, they realise that the answer is "no" to Mr. Anderson's question, "Are there, at present, sufficient general practitioners scattered over the country to give adequate and efficient service to the whole animal population?"

Modern veterinary medicine demands frequent visits to the farm and the establishment of a close personal contact between practitioner and farmer. This can only be achieved economically and effectively if travelling is reduced to a minimum.

To do this there must be an increase in the number of practices, either by the decentralisation of far-flung practices, or by the establishment of new compact ones, in which the day's work will be measured by cases seen rather than miles travelled. Some form of contract scheme would seem essential if the practitioner is to have ready access to the farm, and the financial side must be assured by a reasonable per head charge.

Finally I would say that Professor McCunn's generalisation that work cannot be manufactured is wrong, and that there are still many areas where the presence of a well-trained keen young man will very soon produce a practice which the district will then find itself unable to do without.

Yours faithfully,

18, Douglas Crescent, C. S. Grunsell.
Edinburgh, 12.

DISEASES OF ANIMALS ACTS, 1894 TO 1937, AND AGRICULTURE ACT, 1937 (PART IV).

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange*	Sheep Scab	Swine Fever
May 16th to 31st 1949	8	—	31	—	—	—
Corresponding period in—						
1948	1	—	8	—	—	3
1947	5	—	128	—	3	—
1946	3	1	—	—	—	14
Jan. 1st to May 31st, 1949	84	11	214	—	27	5
Corresponding period in—						
1948	44	—	181	1	26	16
1947	39	83	333	1	22	14
1946	47	24	—	4	34	252

NOTE.—The figures for the current year are approximate only.
* Excluding outbreaks in Army Horses.

Tuberculosis (Attested Herds) Schemes

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at May 31st, 1949, was as follows:—

England	Wales	Scotland	Total (Great Britain)
15,454	13,028	11,515	39,997